BRAIN.

JANUARY, 1884.

Original Articles.

THE RELATIONS OF HYSTERIA WITH THE SCROFULOUS AND THE TUBERCULAR DIATHESIS.

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THE numerous labours which have of late so successfully been directed towards the transformation and completion of neuropathology have all been inspired by an analogous tendency, and have left aside an important aspect of the question.

The symptomatic pictures presented by the various diseases of the nervous system have been especially determined, and thus distinct morbid types have been clearly established. The physiological pathology and pathogeny of these various clinical syndromata have also been unfolded; finally, in many cases, the anatomical lesion upon which the disease depended has been carefully analysed.

It is evident that such was the right method in unravelling the ancient chaos of nervous diseases, and it is already a great service rendered to the physician to establish definitively for him the symptomatology, physiology, and pathological anatomy of the diseases with which he has to grapple.

But beyond this sphere of knowledge there exists a whole group of facts, the knowledge of which is urgently needed by him to complete his diagnosis and establish his therapeusis: it is the etiology. This chapter has yet to be written for the nervous diseases.

In order that an etiology be living, as it were, complete and clinical, it is not sufficient to indicate the immediate cause which has determined this or that accident, such as a more or less violent emotion, as the beginning of a neurosis, or some sexual excesses as the starting-point of a tabes. Unless we go beyond this proximate etiological element we shall create but a trivial neuropathology, and one without practical applications.

This proposition, which has been at all times as the by-word of clinical medicine, will be still more readily at a time like the present, when even acute pneumonia is considered by many as a localised general disease, and when traumatisms—these most typical of local states—are so visibly influenced and dominated by the constitution, or general habit, of the patient. Most physicians recognise nowadays that the local or physical diagnosis in a pulmonary or cardiac disease, important as it certainly is, remains much below what is needed for a complete and practical knowledge of the disease; that it must be completed by a diagnosis of "nature," by an analysis of the general morbid state which governs this lesion, already well defined by our senses armed with the requisite instruments. If this view is true of the circulatory and respiratory system, it is all the more imperative to take it into account with reference to the nervous system.

Placed in presence of a nervous case, the physician must evidently begin with the physiological diagnosis, that is, carefully analyse the symptoms and connect them with the normal functions of the nervous system; he next proceeds to the anatomical diagnosis, determining the presence or absence of a lesion, its position and extent. This part of the problem solved, and all late researches have tended to facilitate this solution, he will be able to label his patient as tabes dorsalis, hysteria, or cerebral softening; but he will not have reached the end of his duty.

There still remains the *nosological* diagnosis; that is, the determination of the general morbid state, of the diathesis of the constitutional disorder which stands behind this lesion or this neurosis. Upon its establishment depends the institution of a complete and rational treatment. Many physicians

accept these views to a certain extent in the case of syphilis, and give their patients the advantage of an antidiathesic medication—but many others decline to entertain them, even with reference to syphilis. At any rate, they are not usually extended to other diatheses, which for us is a mistake.

The fact that we have not as powerful means against rheumatism, scrofula, and other diatheses, as against syphilis, does not imply that we stand disarmed in presence of them. There is an anti-diathesic indication to be fulfilled, and for the fruitful administration of iodine or alkalies it is not indifferent to know whether the disease ascertained is of rheumatic or scrofulous nature.

Neuropathology then requires, like the other branches of pathology, to be completed and enlightened by a thorough nosological study of the causes of the general morbid states.

This preamble was necessary to justify our attempt to study the relationship of hysteria with the scrofulous and tubercular diathesis;[1] and to write, so to speak, a paragraph of this chapter of neuropathology.

Hysteria is a disease of such complexity, tenacity, and peculiarity, that it is often usually considered as a true morbid state, distinct from and irreducible to other disorders. Such is not our view.

Hysteria is a neurosis; but we scarcely can admit of a neurosis except as symptomatic. They point to a peculiar condition of the nervous system which is unknown to us, but which exists—dynamically or anatomically; and this state of the nervous system is in its turn symptomatic of a general disease, or more usually of a diathesis.

Just as chorea is often a manifestation of the rheumatic, and angina pectoris one of the gouty diathesis, thus hysteria is often a manifestation of various diatheses, more particularly of scrofulosis and tuberculosis. Hence when we speak of the relations of hysteria to these diatheses we do not mean that hysterical subjects have frequently tubercles in the lungs, or

[1] It is chiefly tuberculosis which we have in view in this paper: scrofula is accidentally mentioned, in consequence of the indications presented by the two diatheses in certain clinical cases.

that the confusion between them and the scrofulous be more or less easy—this is not at all the aspect of the question we are contemplating.

We mean simply that these diatheses are constitutional states found in a series of generations of the same family, and that among a certain number of subjects of the series they may manifest themselves as neuroses; that especially in certain individuals, children of tuberculous or scrofulous parents, the tuberculosis or scrofulosis may be represented by hysteria only.

In order to grasp this view, we must admit that the diseases known as tuberculosis and scrofulosis are of a diathesic, constitutional nature, frequently manifested by the anatomical lesion designated as tubercular or scrofulous, but not necessarily linked to such lesions.

Tubercles, for me, are not necessary and uniform characteristics of the tubercular diathesis; they occur without it (impaludism, scrofulosis, alcoholism), and there are cases of tuberculosis without tubercles. I cannot develop this proposition here; but I consider it as a demonstrated clinical truth.[1]

Rheumatism may be manifested by chorea, gout by angina pectoris; that is, lesions of the serous or fibrous tissue do not necessarily occur among all rheumatic subjects, nor the uric lesion among all the gouty. In other words, diatheses are not manifested by this or that lesion; they are indissolubly linked to no single anatomical lesion special to them. They may become apparent as neuroses, that is, by syndromata, of which the lesion is not appreciable to our senses.

What we are now saying of rheumatism and gout is true of syphilis and the other diatheses, such as scrofulosis and tuberculosis. It was in order to avoid the similarity between the words "tubercle" and "tuberculosis" that we proposed to call the latter "Laennec's disease," a name which predicates nothing of the anatomical substratum of the disorder, and is therefore not in contradiction with the view we support.

When therefore we say that tuberculosis may become manifested as hysteria, we do not in any way imply that there are

[1] See our article *Diathèse*, in 'Diction. Encycl. des Sci. Méd.'

tubercles among hysterical people. It is not the coincidence of hysteria with pulmonary or other tubercles which we want here to place into light. We want to show that hysteria may be, and often is, a manifestation of the tubercular diathesis; and two cases may present themselves here according as the neurosis will be the only manifestation of the diathesis, or will be continued in the same subject along with other diathesic manifestations, such manifestations bearing to one another certain relations to be studied.

How is the clinical demonstration of such a proposition to be made?

I must say at once that it is not by the direct examination of the patient; it is by the whole pathological history of his life, ascending into its remotest origins; it is by the history of the subject and his family, by the study of the transformation of the disease through generations.

By thus working up into the morbid antecedents of this subject, by following for a long time the ulterior phases of his disease, by analysing the diseases of his ascendants, collaterals, and descendants, one is struck by the relations of intimate affiliation between hysteria and certain diatheses, especially tuberculosis and scrofulosis.

Thus, for instance, in a phthisical family you will see among the children of a tubercular subject one dying of tubercular meningitis, another become an ordinary consumptive, the third seem to escape the diathesis. The last may be neurotic, hypochondriac, hysterical, or lunatic even; but for me he has escaped the hereditary diathesis in appearance only. He is tubercular, like the rest, though he has no tubercles anywhere. It is his neurosis which is the manifestation of his diathesic affection. And what proves it is that in his children the diathesis is still present, and will often reassume its classical type. Son and father of phthisical subjects, he does not interrupt the diathesic chain; only pulmonary phthisis is in him replaced by hysteria. Moreover, he may prove the nature of his neurosis by becoming himself phthisical in the second portion of his life; and then there are often most remarkable phenomena of alternation and equilibration between the two manifestations of the same diathesis, phenomena which

have caused the supposition of an antagonism between hysteria and tuberculosis, when they were merely successive and different manifestations, mutually replacing one another, of the same fundamental diathesis.

This view will shock certain minds at first sight. It however results from the very nature of things, and is not without analogy with other categories of clinical facts.

How often one sees the descendant of rheumatic parents seized with chorea without having previously presented any articular pains! This chorea is truly rheumatic, as will become manifest by the apparition of articular disorder in himself or in his children. No one hesitates in such a case to frame such a diagnosis. The same considerations obtain when we deal with migraine, asthma, and arthritism, and of all this group of clinical syndromata, of which Bouchard has so well brought the intimate relationship into light in his recent work on diseases from slackening of the nutritive processes.

This relation which we wish to establish between hysteria and certain diatheses does not therefore contradict clinical experience, and must not appear *à priori* irrational. Now it is easy to see the practical importance of this kind of study. The consideration of the diathesis standing behind hysteria, and, generally speaking, behind all neuroses, is not a theoretical notion of pure pathology, but a real clinical datum, yielding indications for the therapeusis.

When one reads the recent important works on nervous diseases, one is infallibly struck at the scantiness of the therapeutical chapters. In the case of hysteria, in particular, one multiplies indeed the means of treating the attack, of removing the anæsthesia; single symptoms are pretty well met and corrected. But the groundwork itself, the neurosis, the morbid state? How powerless we seem in many cases! One scarcely attempts, even, to establish a rational mode of handling the factor which keeps under its dependence all the ever-changing scene of manifestations.

And yet it is there that a new and fruitful path lies. As we shall try to establish it later on, the treatment of the manifestation is often useful; the treatment of the nervous system is indispensable; but more than all this is wanted.

The deeper organic cause of all these nervous derangements, the morbid state which is the true disease, has to be grappled with. We only indicate here the ideas which the remainder of this paper will develop and demonstrate. We shall best return to them after giving the facts upon which they rest. But it is indispensable to put forth synthetically at the very outset the doctrine which we wish to enforce. The reading of the cases will thus be more profitable.

We shall not enter upon the history of the question, which would indeed be very short, authors having scarcely yet considered and discussed this view of hysteria—and this, for several reasons. Firstly, not thinking of the relationships, they have not looked for them—and these are things to be sought. Further, such researches are almost impossible with the material supplied by hospitals. The patients who go there come from unknown quarters; we are debarred from every accurate investigation into their heredity. It is only in private practice, where one has an extensive knowledge of one's patients, of their ascendants and descendants, of their whole family, that one can frame such a nosological diagnosis.

The causes of hysteria in hospital patients are also usually too complex, and one may say too external, to allow the putting into clear relief the pathogenetic rôle of a diathesis. Why seek tuberculosis among the ascendants of a Saltpêtrière patient? Her whole life is insufficient to account for the development of the neurosis; whilst among the upper classes, if you find hysteria in a young woman, or even a child, in a family with a calm existence, with regular habits, and no moral over-driving, you will seek more attentively for the hereditary constitutional vice which has struck this patient, and you will easily discover the diathesis, of which the importance becomes immense.

The view which I defend here has therefore been frequently indicated to me by my master and colleague, Professor Combal, who, in his immense private practice, continually finds proofs of the intimate relationship between the neuroses and the diatheses. In the first edition of my work on the Diseases of the Nervous System (1879) I said (vol. ii. p. 555): "The general states may also give rise to hysteria . . . diatheses are

found in some cases, and would be oftener found if better looked for. Thus one often talks of hysteria among the children of a consumptive subject, and rightly. Hysteria is in such the manifestation of the hereditary diathesis, and cases are seen in which this diathesis manifests itself at first only by nervous disturbances, and later by the typical lesions of the lungs, whilst at the same time one often sees the hysterical phenomena diminish and disappear."

In the second edition (1881), I reproduced this quotation (p. 924), and added (p. 1040): "Finally the tubercular diathesis may betray itself by neuroses, hysteria especially, chorea, &c. I have recently seen two cases in which the hysteria seemed to be the manifestation of the tuberculosis. These facts have a great clinical importance, because frequently hysteria is developed in a young woman with a diathesic heredity; the neurosis is treated; it disappears, and then pulmonary accidents, sometimes of the utmost severity, are evolved. Or the hysterical phenomena may persist during the phthisical process, and present a series of compensatory rises and falls. Finally, hysteria may be the only expression of the diathesis, and phthisis reappear only in the succeeding generation." And I quoted in a note an observation from the "Iconographie photographique de la Salpêtrière," and two from the 'Thesis' of Dr. Paris, which will be mentioned again.

The following authors, also of the Montpellier School, have published papers which may be taken in connection with our present subject: Brousse,[1] Largaud,[2] and Mossé.[3]

Most classical authors either say nothing on the subject, or mention our doctrine only to set it aside. Thus in his recent 'Traité des Névroses,' Huchard says (p. 1056): "Some authors have made a mistake in saying that tuberculosis gives rise to hysteria:" further (p. 1073): "Among the diathesic diseases, phthisis has been quoted among the antecedents of hysteria. However, it is not the latter morbid state which is to be incriminated, but only the arthritic diathesis which may give rise to both tuberculosis and hysteria, and the latter is not in any way depending upon the tubercular affection."

[1] 'Gazette des Sci. Méd. Montpellier,' 1881, p. 423.
[2] Thesis No. 23, 1882.
[3] 'Gazette de Montpellier,' 1883.

We shall discuss, in a later paragraph, this theory and several others propounded upon this subject. We must first produce our facts, and prove the relationship; the question of interpretation will come later on.

Our cases will fall into two groups. In the first, hysteria is the only manifestation of the tubercular diathesis. In the second, we shall find simultaneous pulmonary and hysterical manifestations. The two categories will serve to establish etiological relationship between the diathesis and the neurosis. The second alone will allow us to study the reciprocal influence which the two orders of diathesic manifestations appear to exercise upon each other.

I. Observations of Hysteria, with Tubercular Heredity without Pulmonary Manifestations.

Case I. (personal).—*Neuropathic and Diathesic Heredity (Arthritism, and especially Scrofulo-herpetism). Hystero-Epilepsy, beginning at three years of age, with Scrofulo-herpetic Manifestations.*

J. C., a little girl of three years old, was seen by me with Dr. Bouffier, of Cette, who kindly furnished me with the notes of the case.

Neuropathic heredity.—Sister died at 8 or 9 months from meningitis, with violent convulsive attacks; brother died from cerebral accidents, during a laborious dentition, at 13–14 months. Maternal grandmother had severe attacks (epileptic?) and died insane. Mother very nervous. Maternal aunt has had violent attacks after strong emotion (fear of drowning).

Diathesic heredity.—Maternal grandfather has had cancroid of the upper lip. Mother very lymphatic: ganglionic enlargements, with attacks of eczema behind the ears. Maternal uncle: marked scrofulo-herpetic accidents. The sisters and brothers of the mother, says Dr. Bouffier, are all scrofulous, lymphatic, or of slow intellect. They are all below the average mentally, but those whose constitution is the best are the least developed in intelligence." Father lymphatic. Paternal grandmother rheumatic. Paternal grandfather has had rheumatic sciatica of several years' standing.

History of patient.—Carefully followed from birth, owing to cerebral affections of brother and sister, and has often taken bromide against the neuropathic heredity. Has had sometimes lymphatic enlargements of neck, and has had a kind of "mucous fever" lasting three or four weeks.

Last summer she had a great fright from falling into a tub, head foremost, after which she had an eczematous eruption on the face. She got better, but another emotional shock brought about another eruption, but less extensive. Three months after, during a walk with her father, was seized with a vertigo; and another followed two or three days after at the same hour, and under the same conditions. The same phenomena went on repeating themselves for a few weeks every two or three days. The attacks were very short, when, two months after the beginning, they were followed with loss of consciousness and resolution of limbs, and preceded by an aura, with alternative pallor and redness of the face, retraction of thumbs and convulsion of eye-balls. The attacks became more frequent. When I saw her she lost consciousness every afternoon at 4 or 5 o'clock, remained motionless, and as dead for about half an hour. No convulsions. She usually feels the attack coming on, gets into her mother's lap, and moans sometimes for a while. Or she is seized suddenly during her play or her tea. She often resumes, on coming round, her occupations where she had left them. Capricious temper.

I prescribe a continuation of the bromide, general cold ablutions, iodide of potassium and arsenite of soda, alternating with gold and sodium chloride, a series of blisters to the arms, (cod-liver oil is not well borne), and I mention a cure at Uriage for the following summer.

On October 28th, 1882, Dr. Bouffier writes: "The child seems much better. The summer passed without attacks, a few vertiginous attacks like the first were noticed at the beginning of the autumn, but have disappeared since. The temper is better." During the last two springs, she has had crops of small boils on the neck, face, and legs. This autumn she presents disseminated pemphigus bullæ and enlarged cervical glands.

In this case the hysteria is not doubtful, and the scrofulous diathesis is very clear in the heredity, and even in the aspect of the subject. Further, the neurosis develops itself without any of the great moral or physical perturbations which mostly bring about hysteria. The age of the patient and the surroundings in which she lives make, it seems to me, the pathogenic part played by the diathesis more clear and important.

Case II. (personal).—*Scrofulous Diathesis.*—"*Hystérie fruste*" (*incomplete hysteria*) *of a chiefly sudoral nature.*

On Oct. 13th, 1882, a girl of 14 was brought to me, who has menstruated since the age of 11½ years, and who for the last three

days presents extraordinary local sweatings. All the external clothing is soaked round the waist. She has saturated up to twelve napkins in a day; and actually (3 o'clock P. M.) wears the third flannel of the day. She feels at times, and very suddenly, a very violent pain, which constricts the lower part of the thorax, and immediately afterwards she feels herself inundated by an enormous flux of perspiration at the stomach; the liquid flows to the ground. These sudoral attacks have begun three days ago after a violent emotion, having been beaten by her aunt, who frequently ill-treats her. Menses have just appeared.

For the last eighteen months she has suffered from frequent and very loud abdominal noises, and for which she had already consulted me; extraordinarily intense borborygmi, during whole hours, simulating every kind of external sound, and very objectionable to the neighbours. At school she has also had frequent syncopes.

Her father is said to have died from abdominal cancer. The mother, whose conduct was bad, died of consumption when the child was 4 years old.

She has had numerous scrofulous accidents during childhood; disorders of the ears, protracted suppurations, chilblains, impetigo of the scalp, ganglionic enlargements, &c. She still has a deeply scrofulous aspect. Her body is weakly and spare. No thoracic symptoms, except a very slight dulness towards one of the external clavicular regions.

Atropia pills cut short the sudoral crises; after which I institute a chronic treatment with cod-liver oil, iodide of potassium, quinine, and blisters.

The curious (secretory) form of the hysterical manifestations would deserve some remarks if they were not beside our actual object. I shall only insist here upon the fact of the neurosis, together with the scrofulo-tubercular heredity and constitution.

CASE III. (personal).—*Neuropathic and Scrofulo-tubercular Heredity; Scrofulous accidents; Hysteria of a chiefly bulbar form.*

Miss X., aged 27, seen the 27th of June with Dr. Bompaire, who has kindly sent notes to complete the present observation.

Neuropathic heredity. — Paternal grandmother insane; father strange. Paternal uncle "has done foolish things which, without the intervention of the family, would have necessitated his reception into an asylum."

Diathesic heredity.—Mother suffers from marked herpetism. Three sisters; one died young after whooping-cough; another died at 11 years old, from "cerebro-spinal meningitis of rapid evolution (five days);" the third had scrofulous accidents at the time of puberty; abscesses in the inguinal and sternomastoid region: died tubercular at 18 years of age.

History of patient.—Scrofulous aspect; nose eczematous. Menstruation is regular, but began late. Frequent facial erysipelas (six or seven every year) between 18 and 23 years. At about 25, fixed pain about the epiphyses of the sixth and seventh cervical vertebræ; and apparition of fits occurring during the day, without loss of consciousness, beginning with coldness and numbness of extremities, with acceleration of pulse, and elevation of temperature, dilatation of pupils, redness of face, cutaneous hyperæsthesia, great volubility of speech, and convulsive jerks violently shaking the limbs and trunk. These fits occurred chiefly before, during, or after the menstrual periods, which have always been very regular. In the interval of the fits, great nervous mobility, and impressionability. At other times globus, nervous cough, &c.

At the moment of my visit all the phenomena are concentrated in the labio-glosso-laryngeal district; impossibility of swallowing other things than liquids. Some time ago she could not blow out a candle. At times, under the influence of an emotion, or without known cause, arrives the fit, which often begins with coldness of extremities, and is manifested by contraction of lips, cheeks, tongue and pharynx. Some time ago she could not put out her tongue, nor especially keep it out of the mouth. Water applied to the neck excites the fit; the patient cannot wash her neck.

I prescribed, besides the treatment against the hysteria itself, cod-liver oil, chloride of gold and sodium, and the application of a blister or the cautery. This treatment had no great result. "Since your visit," writes Dr. Bompaire, "the fits gradually diminished, then disappeared; then supervened nervous disorders of the pharynx, preventing the patient from swallowing solid or half-liquid substances. A metalloscopic examination seemed to show that zinc plates applied in front of the neck increased the symptoms, whilst gold seem to mitigate them. Several bottles of the auro-sodic chloride have been taken without alleviation, and the patient was sent (end of July, 1881), into the country at an altitude of 900 metres. There she could not go out once. She was seized with a 'nervous fever,' lasting three weeks, a remittent fever during which the pulse oscillated between 110 and 140, and the temperature between 38° and 40° C., with nightly exacerbations. The treatment consisted in the administration of quinine and aconite.

"During the last winter and spring the troubles of deglutition were complicated with an abundant sialorrhœa, the patient using as many as twenty pocket-handkerchiefs in the 24 hours. The saliva came chiefly from the parotid glands. At one time the weakness was so great on account of the deficient feeding, that peptones had to be employed, with good results.

"Since then the family decided to submit her to a quack treatment; and as I refused to conduct it myself, ceased to call me in. At the present time the patient's condition is more satisfactory. She can get up, and take a few drives. The difficulty of swallowing still persists."

Here again we find the double heredity, neuropathic and scrofulo-tubercular. Let us note in the subject the strumous manifestations and the remarkable (bulbar) form of the hysteria.

Case IV. (personal).—*Tubercular Heredity. Rough breathing at the Apices, and frequent Bronchitic attacks. Hysterical fits, Rachialgia, &c.*

Mrs. G., aged 38.—Mother died after parturition, but slowly from consumption. Brother died, from slow consumption also, after the fatigues of the war.

History of patient.—First hysterical fits at 20 years, without apparent cause; grave, sometimes cataleptic; occurred repeatedly during several months. Regular menstruation. Three pregnancies; normal childbirth.

Six years ago she lost her husband, and left her home to look after an old, ill-tempered uncle. Protracted moral sufferings. Attacks occur again during eighteen months or two years, at variable intervals. Cauterisation of the cervix. Grave typhoid fever two years ago, with cerebral symptoms. Good health afterwards. In January 1881, during the financial crisis, she hears suddenly that the fortune of her children has been lost through secret speculations of their trustee; despair. Starts for Avignon, runs about the whole day, and in coming back in the train has a violent hysterical fit. This is the beginning of the actual disease. For several days fever and delirium. Attacks, at first frequent, disappear later on. Then, three or four months afterwards, great attack (with catalepsy). From the beginning until now, violent pains down the rachis, occasionally going through the thorax, preventing the dorsal decubitus. At the same time progressive onslaught of a paraparesis with bilateral anæsthesia. No urinary

nor rectal symptoms, but at various times nervous vomiting. Cannot walk without a stick, does not feel the ground well. Cannot work, owing to pain caused by moving the arms.

On examination (October 1882), violent rachialgia. Impossible to press on the spinous apophyses. Sensations everywhere dull, especially on the left side, on the right breast, and as high as the knee in the right leg, and the elbow in the right arm. Nothing in the chest except some roughness at the apices. Coughs much since a fortnight. Frequent bronchites. Constant cold feeling in the lower extremity; the attack begins with the accentuation and the generalisation of this cold.

I send her to Dr. Regimbeau for applications of static electricity. The first day the very sight of the apparatus produces a violent fit. Afterwards the electricity is well supported. She leaves on the 1st of December, much better. I prescribe a lengthened treatment with the ferro-sodic arseniate.

Here there is no neuropathic heredity observed. But numerous and violent moral excitations have served as occasions to the tubercular diathesis to determine the neurosis.

Case V. (personal).—*Tubercular Heredity. Hystero-Epilepsy.*

Mrs. B., age 32, was sent to me in July 1882, by Dr. Cayla, who kindly supplied some notes to complete the history of this patient.

Heredity.—Father died young of pneumonia. Mother died four years ago of heart-disease. The patient is the eldest of three daughters. One of her two sisters died phthisical at the age of 18, and the other, aged 21, and recently married, is hysterical. Mrs. B. has had three children. The one suffers from a general eczema, resisting every treatment; another died at birth.

History of the patient.—Lymphatic temperament; health good till first child was born. Fourteen years ago had stillborn child, upon which had grave eclamptic symptoms for about twelve hours; after then frequent sick headaches. The violent frontal pains become real seizures, occasionally accompanied with very short loss of consciousness, preceded with vertigo, and followed with loss of memory during a few seconds. No hysteria, globus, nor convulsive phenomena.

Little by little this condition became worse. The attacks become more frequent; they were characterised by a kind of aura, followed with vertigo and loss of consciousness. Feeling returned after a few minutes, but there remained for a few hours a complete

forgetfulness of what had happened, some cephalalgia and muscular pains. Under the influence of fatigue, and menstruation, which had become irregular and insufficient, but few days intervened between the fits. Loss of flesh.

"After the second childbirth, two years ago, and an illness of her daughter, the nervous symptoms increased in intensity. She was seized with vertigo, placed the baby on the ground, without consciousness of that she was doing, went into another room, came back, and awoke astonished to find herself there. In the night she has left her bed, deposited the baby at the top of the stairs, and returned to her home. I saw her one day seized with a fit, of which she announced the invasion; she became slightly pale, contracted her hands and forearms. Then, with closed eyes, she walked to the middle of the room quite naturally, began to undress. Taken back to her arm-chair she came to herself, much astonished to find on her knees the petticoat she was wearing a few minutes before. In a short time she resumed her normal state."

She is weak, anæmic, of a very versatile temper; memory bad, affective feelings dulled.

I prescribed a hydrotherapic treatment at Lafoux, and on her return, a continuation of the cold-water treatment, progressive doses of arseniate of soda in glycerine, with bromide of sodium; and a purgative dose every month or so.

Dr. Cayla wrote to me in October 1882: "She went to Lafoux for a season, when the number and duration of the attacks were temporarily diminished. But on her return home she has not followed the prescribed treatment, notwithstanding the instances of her friends, and her state is much the same. I wished to send her again to Lafoux, but she has not consented."

Though tuberculosis has not been found among the ascendants, it seems to exist in the family, on account of the case of the sister. The facts are then so: of three sisters, one is phthisical, the two others hysterical, and the eczema in the daughter of one of the latter reproduces the classical manifestations of the scrofulo-tubercular manifestations.

CASE VI. (*Second case of Paris, 'On the Hysteria of Little Girls.'* 'Thesis,' Paris, 1883.)—*Tubercular Heredity. Hysteria beginning at 10 years old.*

Heredity.—Mother died phthisical. Father in good health; ten brothers and sisters in good health also; two died of disease unknown to the grandmother who brings the girl.

Nothing noteworthy among the personal antecedents.

History of disease.—Nervous attacks at 10 years of age, then fits of somnambulism. Various painful symptoms; right ovarian pain, frequent sick headaches, anæsthesia of conjunctival and velum palati. Right hemianæsthesia.

We shall call attention to the presence here of a tubercular heredity in the absence of any neuropathic heredity and of any nervous over-excitation in the patient herself. Moreover, hysteria began early, at 10 years, a fact which connects the case with the first, and with the following.

CASE VII. (*Third case of Paris; loc. cit.*)—*Tubercular Heredity. Hysteria breaking out at 9 years of age.*

Heredity.—Mother died phthisical nine years ago.

Personal antecedents.—Ill-treated by step-mother; whooping-cough, measles, and scarlatina.

History of disease.—Nervous attack at 9 years of age, provoked by contrarieties and fears. Syncopes, with forebodings; ovarian pain on the left. Anæsthesia of conjunctivæ and soft palate; other symptoms.

Here again the tubercular heredity and hysteria are distinct, whilst neuropathic heredity does not exist. There has been, however, much ill-treatment, which may have acted as exciting cause to the neurosis. The latter became manifest at a very early age.

Similar phenomena are noted in the next six cases.

CASE VIII. (*Case 12 of Briquet*, p. 61.)—*Tubercular Heredity. Hysteria beginning in early youth.*

V. D. lost her brother from phthisis; has two sisters always ailing. She has been ill-treated by her parents, and from childhood till 29 years she had every nine or ten days a convulsive attack with loss of consciousness, and with all the characters of hysterical attacks. Worry had much to do with the production of these fits. The latter have ceased since, but there has remained until now (37 years), a well-marked general hysterical state. Two pregnancies between the ages of 25 and 30 have not brought about any fresh attacks.

CASE IX. (*Case* 13 *of Briquet*, p. 62.)—*Tubercular Heredity. Hysteria from childhood.*

A. C. has a hysterical mother. Father died phthisical, and one of her sisters died of convulsions. From her earliest childhood she has, when crossed, choking and spasm of the throat. At 7 years, complete hysterical attack; at 16, chorea; at 17, became again subject to hysterical attacks, which persist up to this time.

CASE X. (*Case* 14 *of Briquet*, p. 63.)—*Tubercular Heredity. Hysteria beginning at 5 years of age.*

V. P. lost her mother and sister from consumption; has had her first convulsive attack at 5 years of age, with loss of consciousness and after violent anger. This fit has all the characteristics of hysteria. Fits still persist now, at 24 years of age.

CASE XI. (*Case* 20 *of Briquet.*)—*Double Tubercular Heredity. Hysteria beginning in the 7th year.*

J. D. lost both father and mother from phthisis. Placed suddenly in presence of a dead body when 7 years of age she had a great fright, and was seized on the spot by a convulsive attack, with loss of consciousness. The succeeding attacks occurred at first several times a day, but gradually became less frequent, and disappeared at about 12 years, when menstruation began. Originally these used to be spontaneous, but later occurred only under the influence of some emotion. These seizures differed somewhat from ordinary fits; they were preceded by giddiness and tinnitus; then after a few minutes there was loss of consciousness, and when the patient recovered there was a choking sensation and epigastric constriction, but no crying. Still, notwithstanding this slight anomaly, the general character of the phenomena was too much like hysteria to leave the least doubt. Moreover, she has had in the hospital, ten years afterwards, when 22 years old, a well-marked and complete attack of hysteria, occasioned by a new fright.

CASE XII. (*Case* 20 *of Briquet.*)—*Double Tubercular Heredity. Hysteria beginning at 9 years of age.*

M. D. lost her father and mother, who died very young, from phthisis. When 9 years old she fell into the hands of relations, where her life, hitherto very quiet, became painful. She was ill-treated, and crying constantly, as if suffocated. She soon became subject

to hysterical attacks, characteristic and complete; they were at first frequent, occurring several times a week, and caused by the least contrariety. They are now less frequent (17 years old), but she presents general phenomena of pronounced hysteria.

CASE XIII. (*Case* 31 *of Briquet.*)—*Tubercular Heredity. Hysteria beginning at* 8 *years of age.*

J. C. lost her mother, a brother, and a sister of phthisis. She has always been ill-treated; one day when 8 years old, after having been beaten more severely than usual, she had a complete hysterical attack, and from that time until 10 years of age she had similar seizures almost daily. They then diminished in frequency to increase again at about 15 years, when menstruation set in with some difficulty. Actually the girl, aged 29, has her attacks every week.

CASE XIV. (*Case* 35 *of Briquet.*) — *Tubercular and Neuropathic Heredity. Hysteria since childhood.*

J. D. has a slightly hysterical mother. Father died phthisical. She always was delicate, very impressionable, and much ill-treated by her parents. From childhood she suffers from choking and epigastric oppression every time she is scolded. Since then her health has always been indifferent. At 12 years of age she menstruated. Since about 15 or 16 she became subject to numbness of the limbs and muscular trembling. At 22 she began to have hysterical fits, and at 29 she is still in a general hysteric condition.

All these facts relating to hysteria beginning in childhood seem to me peculiarly important to our argument. The part played by heredity is here much more considerable, and that of external causes much less marked.

We can thus interpret in an especial manner the general numbers of the great statistics of Briquet. On 351 hysterical patients, the health of whose parents, brothers and sisters, has been ascertained with sufficient accuracy, we find: out of 282 fathers, 9 cases of phthisis and 9 of hysteria; of 327 mothers, 13 cases of phthisis and 103 of hysteria; of 338 sisters, 8 cases of phthisis; of 148 brothers, 10 cases of phthisis and scrofula.

No conclusions can be drawn from the crude numbers; but let us establish categories, and take into account the age at which the neurosis was developed. We then have 80

hysterical subjects before 12 years of age, that is, puberty; on 56 fathers, there are 6 phthisical or cancerous; on 78 mothers, 11 phthisical or cancerous (and 28 hysterical); on 81 sisters, 6 phthisical; on 43 brothers, 3 phthisical.

These numbers give per cent.:—

For hysteria in the adult, 3·19 % phthisical fathers; 3·79 % phthisical mothers; and 3·61 % phthisical relatives.

For hysteria in the child, 10·71 % phthisical fathers; 14·86 % phthisical mothers; 13·07 % phthisical relations.

Having attracted the reader's attention to these facts, we resume our series of condensed cases.

CASE XV. (*Case 66 of Briquet*).

L. M., 22 years. Mother died phthisical; sister similarly affected. Delicate and very impressionable till 17 years, when first menstruation occurred; then suppression of menses. Left pleurisy with effusion (5 to 6 months), marked anæmia afterwards. Between 18 and 20 good health, menses regular. At 20, beginning of hysteria, due to great sorrow owing to death of sister. Various pains, convulsive attacks, rhythmical chorea, hyperæsthesia, and a series of obstinate symptoms leaving no doubt as to the diagnosis.

CASE XVI. (Lombard, 'Gazette Médicale,' 1876, p. 187.)—*Tubercular Heredity, but no Neuropathy. Hysteria beginning at the 10th year.*

Heredity.—"This case is one of a boy of 15, whose family I knew many years since, and where there is no epileptic antecedent. The father had good general health, and died from confluent small-pox. The mother is strong, with a good constitution, though somewhat lymphathic. An elder sister has always been well, excepting suppurated cervical glands. Another sister has had frequent erysipelas of the face during many years, and finally a pulmonary consumption, from which she died. Finally, a brother showed the symptoms of tubercular meningitis, which carried him off. Thus a family completely free from nervous disease, but subject to lymphatism and tubercles."

History.—At 10 years of age, fifteen days after a fall on the head, first attack of the evil. Five convulsive seizures between 1871 and 1875; globus hystericus, &c.; hemianæsthesia, and hemihypothermia on the left side; pain to pressure in the right iliac fossa.

The tubercular absence of neuropathic and heredity are well defined by the writer. A fall on the head seems to have been the existing cause of the localisation. Here again the family history is instructive: out of four children, one has suppurated glands, another a pulmonary phthisis, a third a tubercular meningitis, the fourth is hysterical. Why refuse to the neurosis its relationship to the other manifestations of the tubercular diathesis?

CASE XVII. (*Case 4 of Dunant.* Thèse, Paris, 1863.)—*Tubercular Heredity. Hystero-Epilepsy, and signs of Pulmonary Tuberculation.*

Heredity.—Father died consumptive; mother healthy; out of eight brothers and sisters three died phthisical, the others are well.

History.—Weakly constitution with signs of pulmonary tubercles (no other details). Patient appears to have been epileptic before the age of 20. During eleven months the author observes nothing but hysterical fits. Later, he witnesses an epileptic attack. Observation of a series of hystero-epileptic seizures.

The fact of the presence of pulmonary tubercles in this patient might have made us place this observation in the second category; but the absence of details concerning these phenomena made us prefer to include it here.

CASE XVIII. (condensed). (Observ. III. de Chambard, 'Du somnambulisme,' &c., 1861, page 85.)—*Tubercular Heredity. "Grande Hystérie."*

Heredity.—Father became alcoholic and dissolute after the death of his wife, and had vomiting of blood every month. Drunkenness was very prevalent amongst paternal ancestors. "Her mother died of chest disease, at 36 years old. She did not suffer from nervous diseases, no fits, her character was amiable but weak. The grandmother and two maternal aunts have also succumbed to pulmonary phthisis. Marie H. has two sisters: one died 10 days old; the other, aged 29, is alive, but in weak health, very nervous, and liable to frequent outbursts of anger, complaining of various ailments, of continual cardialgia and of leucorrhœa, which may be referred to a chlorotic and nervous state. She has two children, one a feeble-looking boy and an idiot girl. Her husband is entirely alcoholic."

Previous illness.—She had measles at 5 years old; then during

the following five years she presented symptoms of scrofula, which were treated for a long time by cod-liver oil, and she also had a skin eruption. Menstruation occurred at 18 years; periods irregular, with lumbar and abdominal pains, nausea and vomiting. —No self-abuse, probably not even any sexual relation; no bad treatment, no privation.

Present illness.—At 21 or 22, had a nervous attack after some provocation, followed by different ailments in consequence, such as chorea during two days, repeated malaise, incessant vomiting with gastralgia, extreme mental uncertainty, passionate desire to attract attention, to please others, &c. Susceptible to mesmerism, she presents the different phenomena of somnambulism. Later on, rhythmical chorea (developed by imitation), with acute intellectual excitation, ending in true outbursts of mania.

The absence of neurotic heredity nervous overwork is remarkable, compared with the completeness of the tubercular heredity and the hysterical neurosis.

Case XIX. (condensed). (Obs. XII. of Richer, 'Etudes Clin. ou Hystéro-Epil.,' p. 206.)—*Tubercular and Neurotic Heredity. Grande Hystérie.*

Madlle. X. 25 years of age. Mother and grandmother both died at 30 of tubercle. Father died mad in an asylum, after heavy domestic trials.

The first attack of hysteria came on at 19 years of age. Series of severe attacks of the type of hysteria major occurred.

Case XX. (condensed.) (Obs. of Moreau de Tours. Cited by Richer, *loc. cit.* p. 352).—*Tubercular Heredity. Hysteria, attacks of Delirium.*

T., aged 42, shoe stitcher. Father died suddenly at 82. Mother died of chest disease, had been subject to nervous attacks.

No severe illnesses in her youth. Menstruation at 12 years of age, without attacks. Married at 24; had two children, who enjoyed the best health. The attacks came on after being abandoned by her husband; and in the form of delirium.

Case XXI. (condensed). (Obs. I. of the 'Iconogr. photogr. de la Salpêtrière,' vol. iii.)—*Tubercular and Neurotic Heredity. Scrofulous in Infancy. Previous Hysteria.*

W., married, laundress, entered the service of Prof. Charcot at 18, May 16th, 1877. Father insane. Mother, laundress, died four years

ago of chronic affection of the chest (cough, hæmoptysis); sober, nervous attack after annoyance only; was left an orphan at an early age. Nine children, of which five dead, four of convulsions, one of epilepsy (?) at age of 12. Of the survivors, the patient is the eldest; one boy of 18 has no nervous attacks, but has had hæmoptysis; another boy has been lost sight of; lastly, one girl seems subject to attacks of weakness.

Infancy of the patient.—At 22 months, convulsions, in consequence of which she became paralysed, deaf and dumb. Towards age of 7, condition improved. During this period she had outbursts of passion, measles, eczema of the head; chronic ophthalmia, which has left no traces; severe chilblains.

From infancy, fantastic disposition, attacks which became more marked at 12. Syncopal attacks; then in different forms, hemianæsthesia, &c.

CASE XXII. (condensed). (S. H. Serre. Cited by Mossé, 'Gaz. Hebdom. Sc. Méd. de Montpellier,' 1883.)—*Tubercular Heredity. Local Hysteria.*

Mother lymphatic. Hereditary antecedents, tubercular, on the paternal side. Age 19. Hysterical contracture of traumatic origin. Disappears suddenly, then reappears. Transitory aphasia. Various nervous ailments.

CASE XXIII. (personal).—*Scrofulous Diathesis* (Hereditary and personal). *Grande Hystérie developed in a Child.*

December 5th, 1882.—Little girl of 10 from Bize, sent to me by her medical attendant, Dr. Agostini, to whose kindness I am indebted for most of the following details.

Antecedents.—Mother nervous, having had an impetigo of the scalp during three years; at the age of 7, after vaccination, had for some time enlarged glands, and frequent eruptions on the nose. The maternal uncle presents the same features of scrofulous diathesis.

Present history.—Began with a kind of chorea; some disordered movements of small amplitude limited to the right upper limb; some slight convulsions in the muscles of the face. At times hiccups lasting sometimes for hours, accompanied by shrieking, which could be heard in the street. After unsuccessful use of various remedies (assafœtida, chloroform, chloral, compression of the phrenic), sulphate of quinine made the attacks disappear; however, they reappeared after some weeks' respite, but were always subdued

by the sulphate of quinine. The spasm ceased five or six months before my first examination, but then occurred suddenly a partial paralysis of the right lower limb. The child could no longer walk without the aid of a stick, and complained of pain in the corresponding knee, which was not, however, either red, hot, or swollen. During that time the choreic movements had nearly disappeared. After two months' duration the paralysis gradually decreased, and the day it disappeared completely the choreic movements returned with increased severity. "The alternation between the paralytic and the convulsive phenomena was not altogether complete, but almost so." Some time after, fresh convulsions appeared of the same kind as the first, but limited this time to the stomach; fruitless efforts of vomiting, like a person vomiting with an empty stomach, and this five or six times a day, sometimes more. The appetite, capricious in the intervals between the attacks, then becomes very keen, a kind of boulimia. The sulphate of quinine suppressed these phenomena, which reappeared a month afterwards; the same remedy again succeeded, but then failed the third or fourth time. The hydro-bromate of quinine then succeeded, to fail again. Rivière's mixture also succeeded at times—she was treated at one time simultaneously with iodide and bromide of potassium, and cold lotions, douches to the vertebral column, cinchona, arseniate of soda, iron, and sulphur baths.

After examination, I diagnosed hysteria of a scrofulous nature (predominance of the choreic phenomena) and I prescribed—sulphur and arsenical baths in turn (later on, wet packing, chloride of gold and sodium, and extract of valerian), a belladonna plaster at the pit of the stomach, and the cautery or repeated blisters on the arm. In the spring she was to be sent to a hydropathic establishment or to a mineral bath.

On her return to Montpellier, and before beginning any treatment, the spasms of the stomach ceased, and were replaced by attacks which came on in the night nearly always at the same time, towards 4 or 5 in the morning, consisting of a profound sleep from which the child is awakened with difficulty, and during which she snores considerably. If awakened, she rises up quite astonished, and the snoring ceases, but she complains of headache and giddiness. This giddiness returns even during the day. If left in bed, she is immediately seized with an irresistible desire to sleep, and the snoring attacks return at once. During this time she yawns very often, sneezes, and only awakens after having been shaken. The valerianate of quinine given by Dr. Agostini, influences the attacks and retards them.

These new phenomena confirm me in my diagnosis; I keep

fundamentally to the same treatment, advising the valerianate of ammonia and the magnet of Charcot.

The 9th of May, 1883, Dr. Agostini wrote to me:—"At the same time I wish to ask you to which watering-place it would be best to send the little girl suffering from hysterical chorea that you saw this winter, and who is going on pretty well on the treatment prescribed. Since you saw her she gesticulates less, but the gastric crises continue in spite of everything; first she has hiccups for two or three days, then nausea and vomiting, then the sleeping attacks, then she again is free from anything; but she never remains a whole week without having something. It is a true visceral chorea."

On the 25th of June the child was brought to me. A fortnight before, a scrofulous kerato-conjunctivitis showed itself in the left eye; since then all the other attacks disappeared. I prescribed a course at Lafoux (hydropathic establishment), then a course at Uriage (sulphur and sodic chloride waters). From this latter place Dr. Teulon Valio (to whose care I had entrusted the patient) wrote to me on the 23rd of July: "The hydro-mineral treatment has been adopted with a view to influence the scrofulous diathesis and to modify the symptomatic manifestations: baths of gradually increased strength and duration, and alterative and purgative waters to drink. I should have kept to these purgative waters if the parents of the patient had not drawn my attention to the fact that the purgatives were always followed by a return of the nervous attacks. The four first baths passed off well. After the fourth, a sleeping crisis came on. To prevent their return, and relying on the comparatively satisfactory results obtained at Lafoux by moderate douches, I prescribed a very weak Scotch douche.

"This treatment not having produced the expected result, I had recourse to baths which act with more energy on the entire system. Since yesterday the sleeping attacks have not returned; but have been replaced by a fit of hiccup. This attack was far less violent than the preceding ones, but was followed by a very marked weakness of the left inferior limb. The patient can only walk supported by her friends, and complains of a pain in the tibio-femoral joint. This pain is not increased either by pressure or by flexion or extension; walking only brings it on. There is not the least febrile reaction." The cure was continued very successfully, *everything* disappeared, and I found a great improvement in the patient when I saw her the following 8th of August.

The crises, however, reappeared in the form of hiccup, vomiting, sleeping or true somnambulism. With regard to this latter species of phenomena, Dr. Agostini writes on the 22nd of October:

"If the sleeping crises disappear for a few days, we are sure to see the return of the hiccups, or of the nausea with or without vomiting. These nervous sleeping crises generally happen in the morning, but can appear at any hour of the day, and last, as the case may be, half an hour, an hour, or more. Two days ago a sleeping crisis lasted seven hours, from which it was impossible to awaken her, in spite of all the means used. These attacks, which before were simple crises of nervous sleep, during which the child remained quietly in bed, are now crises of somnambulism. Although the child's eyes are shut, she sees and hears everything during the crises, knows who comes into the room by their step and walk. If anything unpleasant is said to her, or if they threaten to wet her, or make her smell something, she gets angry, and pushes everything violently away that is offered her. She replies by signs to all questions, and if she wants anything, asks for it by gesture: if she wants to drink, she puts her finger to her lips as if in the act of sucking, and if not understood, becomes irritated, gets up, and leads the father or mother to the sideboard or cupboard where the things she wants are, and always with the eyes shut. She is able to go through the whole house, I do not say quite alone, because they do not allow her to walk alone, but with help, and she directs the way wherever she wishes to go. During the crises she even sometimes amuses herself with a little dog, which she makes jump over a stick resting on the cross-bars of two chairs opposite each other, and according as she wishes the dog to jump higher or lower, she moves the stick to the lower or higher bars of the chairs; and that with eyes always shut spasmodically. During the whole attack she cannot talk; but towards the end she speaks, although not yet recovered from her state of somnambulism.

"When the crisis is over, the child remembers nothing of what she has said or done in her sleep, and is much surprised at everything told her."

I advised them to try artificial hypnotism, and above all a complete course of hydrotherapy and statical electricity, carried out, if possible, away from the patient's family.

This case is remarkable for the clear manifestation of the "grande hystérie,"—without convulsive attacks, however—on a basis manifestly of scrofulous heredity.

CASE XXIV. (personal).—*Hysteria* (*Polyuria, incomplete Exophthalmic Goître*).—*Tuberculous Heredity.*

Madame G. was sent to me by Dr. Dargelos of Aix, September 27th, 1883, aged 42. Her mother died young of a long illness (of the liver it was said); two relations (uncle and aunt) died tuberculous. An uncle died of diabetes; another of heart-disease.

Menstruation at 10 years of age; for the last four or five months, periods have been at shorter intervals; delicate health; very impressionable; lively imagination. Ten years ago, after violent emotion, attacks of hysteria (unconsciousness, loss of words, and severe convulsions, "grands mouvements;" ending in weeping), every day for a month; they began at a monthly period (accidental suppression), and ceased at the following period. Frequent cephalalgia, almost constant, very violent occasionally. In August 1883 two new attacks; at this time a great diminution of strength, pruritus vulvæ intolerable, intense thirst and considerable polyuria, seven or eight times in the twenty-four hours; no sugar, no albumen; density 1004 to 1006. Further, at the time of my examination, very decided swelling of the thyroid body, particularly the left lobe; no exophthalmia; frequent and violent palpitation.

I prescribed hydrotherapy, bromide of potassium, iodine ointment, and tonics.

In spite of the absence of exophthalmia, the symptoms, we think, are those of (incomplete) Graves' disease, which is well known in relation to hysteria. Here it is one of the manifestations of severe neurosis, very marked; and on the other hand tuberculosis is present; only it is mixed with other hereditary taints, which makes the case less conclusive.

CASE XXV. (personal).—*Tuberculous Heredity. Hysteria.* (*Neuropathic condition.*)

M. P. of Marseilles came to consult me on Nov. 1st, 1883, age 35.

Heredity.—Slightly neuropathic in his mother and maternal grandfather. Father and brother died tuberculous. He himself up to 5 or 6 years old was, as he said, "full of humours;" wets his bed at night up to 15 years. Masturbation; venereal excesses, no syphilis. Marries at 25 a woman, who died of chest disease a year

ago. Four children; two died very young, one of the survivors weak.

His illness is essentially characterised by sensations of constriction of the throat (globus), which occurred after the onset of frequent seminal losses, and pain and weakness in the loins. General nervous condition very marked; incessant distraction; continuous work quite impossible; head weary; imagination constantly at work. No memory, &c.; has taken chiefly bromide of potassium, about 20 grammes a day.

I advised him to leave off the bromide, and ordered hydrotherapy, arsenic in glycerine, and the constant current along the spinal column.

I admit that here the hysteria is incomplete, many would call it simply neurasthenia, all these neuroses, however, belong to the same family. The tuberculous heredity is very marked.

Here we conclude the observations of our first group. The eight personal observations, and the seventeen borrowed from various authors (in a rapid review of the authorities), seem to me to prove that tuberculous or scrofulous heredity is more frequent in hysteria than is commonly believed. I mean hysteria of the usual type, and which has not any extraordinary development.

The family histories which we have described tend to show that hysteria may probably represent, in some members, the diathesis which the more typical phthisis shows in others.

Thus, from my point of view, the doctrine is exemplified in cases like observation V, where, of three sisters, one is phthisical, and the other two hysterical; the daughter of one of these latter having already the classical symptoms of the scrofulous or tuberculous diathesis. This case cannot be ascribed to atavism. The chain of the diathesis has not been broken; it only is represented in one generation by hysteria, instead of by some form of chronic pneumonia.

The family history of observation XVI is just as instructive. Of four children, one has suppurating glands; another, pulmonary phthisis; a third, tubercular meningitis; and the fourth, hysteria. Most medical men consulted for the first three would declare the unity of the scrofulous-tuberculous diathesis, and will make an exception for the fourth. There is no good

reason why the same argument should not be used and the same scrofulo-tuberculous nature be asserted for the nervous hysteria itself.

We shall complete the exposition of the doctrine and its applications, after having given the observations of our second group. Up till now we have noted nothing in the thorax of our hysterical cases; already, however, we have seen in some of them scrofulous manifestations coinciding, or oftener alternating, with the nervous. In the following observations there exist in the same subject hysteria and pulmonary tubercle, so that one can study the relations between the two forms of manifestations of the common primary diathesis.

(*To be continued.*)

ON POSTERIOR SPINAL SCLEROSIS, CONSECUTIVE TO DISEASE OF BLOOD-VESSELS.

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(MICROSCOPICAL EXAMINATIONS OF THE CORD, BY W. BEVAN LEWIS.)

TIME does not appear to have done much towards clearing up the difficulty of deciding a question which really occupies the very threshold of enquiry into the etiology of tabes dorsalis. It is generally though not, I think, universally, agreed that in this disease we have to do with an irritative lesion of the spinal cord, not a simple atrophy. The question, however, may still be asked, as it was put by Charcot ten years ago:[1] "What is the primary seat of this irritative lesion? Is it the neuroglia? Is it, on the contrary, the nervous element?" He goes on, it will be remembered, to say: "I acknowledge that, considering how the change remains confined systematically, in a way, to the area of the posterior fasciculi, the borders of which it does not spread beyond unless in exceptional circumstances, I cannot help inclining strongly to the second hypothesis. The parenchymatous irritation would then be the initial fact; the interstitial irritation would be secondary."

The remark is made in connection with systematic, fasciculated sclerosis, such as is found in tabes dorsalis, and in primary sclerosis of the antero-lateral columns. As regards disseminated sclerosis, Charcot speaks more positively. In his judgment "the multiplication of nuclei, and the concomitant hyperplasm of the reticulated fibres of the neuroglia, undoubtedly constitute the

[1] 'Leçons sur les maladies du système nerveux,' 11e serie, 1er fascicule, p. 6.

initial, fundamental fact and necessary antecedent; the degenerative atrophy of the nerve elements is consecutive and secondary." The hyperplasia of the walls of the blood-vessels, he goes on to say, plays merely an accessory part. In the affection of the neuroglia he finds all the characteristics of "formative irritation." Disseminated sclerosis is a primary and multilocular chronic interstitial myelitis or encephalitis.

So that, according to this view, the relation in point of time is exactly reversed in the two conditions. In the one case (fasciculated sclerosis) it is the nervous element proper which first shows "irritative lesion;" in the other (disseminated or insular sclerosis), the connective tissue. In spite of this fundamental difference in the process, the results appear to be the same. As regards the histological characters, grey induration in locomotor ataxy, according to Charcot, differs in nothing essential from what it is in disseminated sclerosis, or in symmetrical sclerosis of the lateral columns.

Wilks and Moxon, I observe, differing in this respect from Charcot, draw a distinction between "sclerosis" and "grey degeneration,"[1] because they do not find the same microscopic appearances in the "insular" as in the "ribbon-like" form of sclerosis. In the "insular" form, especially in the brain, they meet with numerous granule masses which they have not generally found in the grey degeneration of posterior columns, "whilst this," they say "is softer." On the other hand, they agree with Charcot in believing that, whilst the change originates in the neuroglia in insular sclerosis, it begins in the nervous elements themselves in the grey induration of tabes. Bastian[2] remarks that "the patches of sclerosis which constitute the anatomical basis of insular or disseminated sclerosis do not differ in their essential nature, or in their appearance (macroscopic or microscopic), from the similar overgrowths of the neuroglia that occur in locomotor ataxy and primary lateral sclerosis." If I do not misunderstand him, Bastian believes that in locomotor ataxy, as in disseminated sclerosis,

[1] 'Pathological Anatomy,' 2nd ed., 1875, p. 250.

[2] Quain's 'Dictionary of Medicine,' article "Spinal Cord, Special Diseases of," 1882.

the change in the nerve elements proper follows the overgrowth of the neuroglia. As regards the process of sclerosis, he asks: "Does it begin in the connective tissue elements themselves? or is there some primary change in the small vessels (possibly of the nature of endarteritis) leading to obstructions and a sequential overgrowth of the neuroglia?"

In another place[1] I have described a case which in its developed form was not distinguishable by any peculiarities from an ordinary example of tabes dorsalis, yet had manifestly travelled to that condition through a phase of meningo-myelitis, and I suggested as probable that the inflammatory action might have attacked the connective tissue of the cord through the channel of the inflamed pia mater. The case which I am about to bring under observation is one which, I think, tends strongly to confirm the probability that such a process is, at all events, an occasional mode by which tabes dorsalis is produced. The case is an important one for several reasons, but principally because, owing to its fatal termination, it has admitted of complete examination, and I have had the great advantage of receiving from Dr. Bevan Lewis, who was kind enough to undertake the task, an admirably detailed account of the microscopical appearances in the cord, basal ganglia, and a portion of sciatic nerve. Mr. Nettleship also has been so good as to present me with a valuable report of the changes in the optic nerve, of which I am very glad to avail myself. Deferring for the moment further consideration of the general question, I will now proceed to describe the case.

Henry L——, æt. 39, unmarried, was admitted into the National Hospital for the Paralysed and Epileptic, under my care, on October 13th, 1881, when the following notes were taken by Dr. Beevor, then Resident Medical Officer.

The patient has been a house-painter for the last seventeen years. He has never suffered from lead-poisoning, colic, or wrist-drop, and there is no definite blue line on the gums. He has not been a hard drinker.

In his family history there is no paralysis or fits. His father's sister was insane. His mother died of consumption.

He has never had scarlatina or rheumatic fever; has twice

[1] 'Clinical Lectures on Diseases of the Nervous System,' 1882, p. 207.

had gonorrhœa, and once, four years ago, had a chancre, which was not, according to his account, followed by eruption on the skin, or sore throat.

He dates his present illness from twenty-two months ago, when he caught cold, from having been much exposed during the winter. His illness began with tottering and weakness of the legs, which used to give way with him, and throw him down. He found that he had especial difficulty in walking in the dark. At the same time he had numbness and loss of sensation in the legs, and also some numbness in the ends of the fingers. He has not been able to walk by himself for the last twelve months.

For the last eighteen months he has suffered from shooting-pains, especially in the knees and thick part of the thighs; less in the shins and ankles. He has never had shooting-pains in the head, trunk, or arms. For about a twelvemonth past he has experienced the feeling of a tight pair of stays compressing the lower part of his trunk, together with a heavy pressure all over his head.

His sight began to be affected two years ago, when, as he describes it, his eyes became dim. Two months ago he began to see things double, and then gradually he lost sight in the left eye. He had a blow on the left ear about four years ago, and lost his hearing on that side.

He has never had any attacks of vomiting or pain in the stomach. For the last twelve months he has had some difficulty in using his hands, and has not been able to write during this period. His urine, for the last six months, has been apt to run from him on any exertion, and he has not always been able to pass it on voluntary effort. He has not suffered from nocturnal emissions; his sexual desire has diminished.

Patient is to all appearance a strong healthy-looking man. His lower limbs are well developed, and are not at all wasted.

He cannot walk or even stand without help, and is unable, as he lies, to lift either foot off the bed, though in this posture he can flex his knees, and can also flex and extend the ankle-joints. In his judgment the left leg is stronger than

the right. With assistance he can just manage to stand on his feet. The arms are not wasted, and are indeed well-developed. When his eyes are closed, he has great difficulty in touching the tip of his nose with either hand. The grasp of the right hand measures 25° by the dynamometer, that of the left, 50°.

The patellar tendon-reflex is absent on both sides; there is no ankle-clonus. Direct percussion of the vasti interni causes contraction of the muscle, which is well marked. Percussion of the tibialis anticus produces contraction more readily than normal.

The plantar reflex is obtained after a delay, which is equal on the two sides. The abdominal and epigastric reflexes are also obtained on each side. The cutaneous sensibility of the legs is much affected. He cannot feel tactile impressions from the knees downwards, but can localise correctly sharp pricks with a pin. There is, however, a delay of from four to five seconds in the perception of pin-pricks in both soles of the feet. In his arms he usually succeeds in localising tactile impressions. With the left eye he cannot tell light from darkness. With the right he can read No. 14 Nettleship with difficulty at two feet.

The left eye, by the ophthalmoscope, shows the characteristic signs of advanced atrophy; the right is unduly pale.

A watch is heard at a distance of three feet with the right ear, not at all with the left. A tuning-fork touching the teeth is heard in the right ear, not in the left.

The heart-sounds number ninety-six in the minute, and are indistinctly heard. There does not appear to be any murmur. The urine contains no albumen. The right pupil measures 4·5 mm., the left 5 mm. Neither reacts to light; each contracts during accommodation.

Under treatment with iodide of potassium some improvement was noted in this man's condition when he had been in the hospital a couple of months. The pains were described as not being so bad or so frequent, and there was a little more power in the control of the bladder. But towards the end of January he was attacked with tonsillitis, which was accompanied by prostration, from which he never rallied. Retching

and vomiting, accompanied by "screwing" pain in the abdomen, began in the beginning of February, and continued with scarcely any intermission until his death. After the attack of tonsillitis, his legs became quite powerless—the urine dribbled away unconsciously, and he entirely lost control over the bowel. The sight of the right eye became worse. (He was quite blind with the left.) There was a tendency to formation of bed-sores in various parts of the lower extremities, but, with attention, this did not increase. For about a fortnight before his death the vomiting was constant and no food was kept down. The pains in the legs continued to attack him. Nutritive enemata were regularly administered, but were not retained. The greatest relief that he obtained (though this was but small) was from hypodermic injections of morphia. He died on March 6th, 1882.

According to his own account, the duration of this patient's illness from first to last was twenty-seven months. At the autopsy, which was made by Dr. Coxwell, the body was found somewhat emaciated. There were no bed-sores.

On removal of the vertebral spines and laminæ the dura mater was found to be distended by a considerable excess of sub-arachnoid fluid. The dura appeared somewhat hyperæmic, but this was possibly post mortem. No inflammatory exsudation was visible. On slitting open the dura mater from below upwards, numerous (about 20) small round calcareous bodies were remarked. These were about one or two centimetres in diameter, flaky in appearance; some circular, others more irregular in outline and they were attached to the parietal layer of the arachnoid. The soft membranes were thickened and adherent generally.

The posterior columns of the cord appeared grey to the eye. (The spinal cord and mesocephale were sent to Dr. Bevan Lewis for microscopical examination.) The brain appeared healthy. The lungs were œdematous, but crepitant, the heart healthy. As regards the kidneys, in each the capsule peeled readily, and the cortex appeared somewhat pale. On the surface of the right kidney there were numerous minute abscesses, the size of a hemp-seed and smaller; the infundibula were much dilated and the papillæ flattened. The ureter was greatly

dilated, as also was the pelvis of the kidney, which was lined with greenish pus.[1]

In the left kidney the pelvis was dilated and injected. It contained much fluid pus. The papillæ were greatly flattened.

The walls of the bladder were much thickened, the increase being due to hypertrophy of muscular substance. There was no ulceration of the mucous membrane.

There was atrophy of the intrinsic muscles of the thumb in each hand.

REPORT OF MICROSCOPICAL EXAMINATION OF THE SPINAL CORD.

BY W. BEVAN LEWIS.

THE spinal cord is diseased throughout the whole length of its posterior columns, the dorsal region having suffered most profoundly, the lumbar region implicated to a minor degree, whilst the cervical portion is the least affected by the lesion. The dorsal cord is specially notable for its small size, its shrunken aspect, and a twisted or distorted contour of its upper half, due to disease of its central tracts; the cervical and lumbar regions are less perceptibly atrophied.

The appearance of the diseased columns in transverse sections of all regions of the cord is to naked-eye examination that of grey degeneration, the chrome coloration being much deeper in the healthy columns than in the diseased tracts, which latter become translucent in glycerine as contrasted with the opacity presented by the healthy medullated columns.

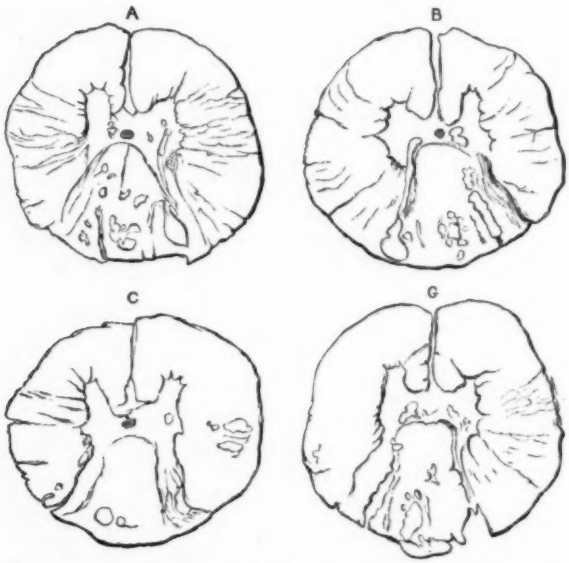
Sections could be obtained with ease from the cervical and lumbar regions, but the dorsal cord, especially in its upper half, was so degenerated and otherwise altered, that it was only with extreme difficulty that tolerably satisfactory sections could be secured.

With low power objectives all stained sections alike exhibit extensive sclerosis of the posterior columns, both the columns of Goll and Burdach being deeply involved, and the former throughout their whole vertical extent. As will be more minutely particularised in describing the regional distribution of the lesions, the degenerative process had extended from the posterior radicular zones as far as the posterior nerve-roots, and, sweeping

[1] The pyelitis was evidently secondary to the disease of the bladder.

across them, had invaded the direct cerebellar tract, and in many cases the crossed pyramidal tract.

In those regions stamped by the greatest intensity of morbid change the anterior cornua had invariably suffered, the various groups of cells being either greatly wasted or wholly absent, and the hemispheric distribution of the lesion was such that, whilst one horn might be entirely free from disease, the opposite cornu might be found shrunken to one half its proper dimensions.



A, B, C, G. Channelling of posterior columns and cornua by diseased vessels in the dorsal region of the cord.
C. Implication of lateral columns in the dorsal region.

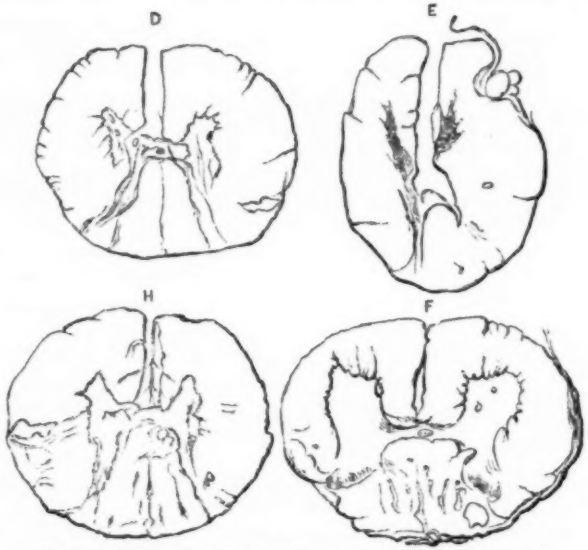
All the structures invaded by the degenerative process exhibit a deeper staining by aniline and other dyes than does the healthy tissue, whilst picric-acid preparations show the characteristic *translucent* aspect of sclerosed nerve tracts.

Even with low power objectives these areas exhibit an apparent remarkable degree of vascularity, the vessels being extremely numerous and dilated, their coats thickened and diseased. In fact, the wide-stretched orifices of these numerous vessels, with their peculiarly thickened walls, was a most notable feature, as

they crowd the field in all directions, invariably forming the centre of a patch of fasciculated sclerosis.

In the *dorsal region* of the cord were first met peculiar *pin-hole cavities*, usually circular or oval in contour, with sharply defined margin, extending down along the course of the medullated strands, or horizontally disposed as elongated excavations or channels directed from the peripheral to the central aspect of these columns.

Such cavities look as though *punched out* of the tissue, and on



F. Channelling of posterior columns and cornua by diseased vessels in the lumbar region of the cord.
D, H. Implication of lateral columns in the dorsal region.
D. Lesions of commissure in the dorsal region, exhibiting also atrophy of anterior cornua.
E. Shrinking of the dorsal region of the cord (upper third).

cutting down through the dorsal region they become more and more numerous, but almost invariably limited to the posterior columns. Occasionally a large cyst-like cavity would be met with, measuring 1·5 mm. to 2 mm. in diameter, or a linear series of such in moniliform arrangement would be seen running parallel to the posterior cornu of one hemisphere along its radicular aspect (Fig. G). Still lower in the dorsal cord such a cavity would be seen in the caput cornu, or at its base, on one occasion through the commissural connections of the anterior horn, and also in the

direct cerebellar tract of one side of the cord (Fig. B, D.). The contents of the cavities were invariably lost in making transverse sections, and their nature was only satisfactorily determined by means of vertical sections through the columns. In such vertical sections diseased vessels were seen in large numbers passing upwards through the sclerosed tracts with many *ampullar* enlargements along their course—these enlargements occupying the spaces above referred to—which are therefore identified as unnaturally expanded perivascular channels. These nodose and moniliform enlargements along the vessels were not aneurismal, but were due to vast accumulations of large nucleated cells which occupied the perivascular sheath of the vessel. The actual calibre of the vessel was sometimes dilated; at other times it appeared constricted by the encircling mass of cells; whilst occasionally, although *rarely*, a genuine aneurismal condition of the vessel had been produced along its diseased tunics. The moniliform channels were occupied by a corresponding moniliform series of such cellular outgrowths upon the vascular walls, and thus it appeared that the whole dorsal region of the cord had been subjected to an invasion by an extensive peri-arterial affection, and its medullated strands exposed to destructive pressure from these nodular outgrowths on the vessels.

The investing membranes of the cord were notably thickened, closely adherent to the surface; their vessels tortuous, enlarged, and similarly implicated where they lie in proximity to the posterior columns. These diseased vessels from the pia mater pass into the substance of the cord through tracts of sclerosed nerve tissue, which lie on either side of them, and more or less definitely appear to follow their various ramifications. The vessels invariably exhibit thickening of their coats, proliferation of their cellular elements, and a peculiar vitreous lustre; whilst the cells crowding the perivascular lymph sheath have each one large nucleus, nearly as large as its investing cell, readily stained by carmine and aniline, and apparently derived directly from proliferation of the endothelial elements of the lymphatic sheath. The sheath throughout its whole course contains large numbers of such elements, but they chiefly congregate in nodular heaps around the vessel as above described.

It appears to me that the peri-arteritis, originating in the membranes and spreading inwards along the nutrient supply of the posterior columns, plays the chief rôle in the production of the lesions to which the medullated strands have succumbed.

As to this presumably *secondary* lesion induced in the nerve elements, the following points were noted.

In all vertical sections of the posterior columns are observed vast numbers of oval-shaped bodies, arranged more or less in linear series along the direction of the medullated fibres. These oval bodies are coarsely granular, and strongly stained of a deep yellow or yellowish-brown tint, and when treated by osmic acid, invariably show a *darkened nuclear-like body* in the midst of the granular elements. The medullated sheath is itself peculiarly affected by *large and irregularly distributed varicosities*—scarcely a fibre was detected unchanged in some part of its course in this manner. The axis cylinder does not appear enlarged, but often contorted, twisted out of position by the changes undergone in the medullated sheath, or obscured in part of its course, and often entirely unrecognised. These diseased medullated fibres are separated by abundance of fine fibrillated tissue, specially well seen in vertical sections.

The moniliform varicosities of the nerve fibres were due to segmentation of its medulla through proliferation of its interannular cells; these interannular cells being represented together with cellular elements around the fibre by the ovoid granular bodies containing deep-stained fragments of myeline (on osmic-acid treatment). In all respects the changes here observed were identical with those lesions seen, after section of a nerve-trunk, in its peripheral segment.

Such was the nature of the lesions found in every morbid portion of the cord, whether the white medullated columns or the grey matter was the subject of examination.

The Cervical Cord.

a. Posterior Columns.—Viewed by low powers, the whole of these columns are found more deeply stained than other parts, the deepened tint continued backwards along the posterior horns across the roots of the spinal nerves, and beyond them to the direct cerebellar tract. An apparently healthy portion of tissue intervenes between the posterior horns and the diseased part of the radicular zones. The columns of Goll are invaded symmetrically on each side, the innermost portion, however, near the posterior commissure, showing comparative immunity from fibroid change and atrophy of nerve elements. The transverse sections are characterised by the notable inequality of the nerve elements. Greatly enlarged medullated tubuli are scattered here and there over the field, whilst intervening fibres are atrophied, or almost replaced by finely punctated fibrous tissue. The large swollen tubuli represent sections across the varicosity of a fibre—the

smaller elements being sections across the more constricted parts of the fibre.

The more deeply stained diseased areas owe their intensity of colour not only to increased connective, but to the vast number of abnormally large and thickened vessels which form a notable feature both in the columns of Goll and Burdach. The internal and external posterior arteries (Ross), together with the arteries of the commissure, and the radicles for the posterior horns, have all suffered from the inflammatory changes in their lymphatic sheaths, and the internal posterior nutrient branch is especially implicated; it is enlarged, and takes a tortuous course as if elongated, through its sheath, which is stretched tense and smooth over the vast accumulations of nucleated cells crowding the walls of the vessels. In no case has hæmorrhage occurred into the sheath, or into the substance of the cord.

From near the level of the sixth cervical nerves downwards the destructive lesions are most severe, but the columns of Burdach are less involved at the level of the fifth cervical nerve, and from this point upwards the extent of morbid action is greatly diminished, so that at the level of the third nerve two narrow ribbons of sclerosed tissue alone are seen, one in each radicular zone, separated by a fairly wide tract of healthy tissue from posterior cornu and commissure.

The posterior roots of the spinal nerves, moreover, at this site issue from the cornua in clearly defined healthy bundles, whilst lower down the cervical cord these roots pass through sclerosed tissue extending into the cerebellar tracts.

β. Lateral Columns.—As before stated, the *direct cerebellar columns* are involved by extension of the lesion along the margin of the posterior columns outwards across the posterior roots. The entire area of these fibres is implicated in both hemispheres of the cord, the morbid change mapping them out most distinctly from the crossed pyramidal tract. The *crossed pyramidal* and other tracts of the lateral columns are in this region perfectly healthy.

γ. Anterior Columns and Root Zone.—The columns of Turck are healthy. The anterior root zone shows a morbid change, limited to one hemisphere, and most distinctly apparent at the level of the fifth or sixth cervical nerves. The hemisphere alluded to has a rounded contour in contrast to the broad sweep of the opposite and more healthy segment. This alteration is to be attributed to central changes in the corresponding cornu, as will be hereafter seen. The anterior roots in this altered segment, however, when contrasted with the opposite nerves under a low objective, are seen to be far less defined and sharp in the contour

of the individual fibres. With higher powers they are seen to have very tortuous axis cylinders, which, although not swollen in any part of their course, are very indistinct, as though interrupted by the *varicose enlargements of the medulla* of the sheath, which are also apparent here. Occasionally, though rarely, a similar change is met with in the *descending medullated strands* of the anterior root-zone in this hemisphere, for here and there may be seen in these columns an enormous pear-shaped swelling of the medullated sheath, into which plunges an axis cylinder, to be lost from view in its interior, again traced further on at a subsequent constriction of the medullated sheath.

δ. *Anterior Cornua.*—An extension of the lesion was traced into the anterior horns at the level of the fourth and fifth cervical nerves, implicating one hemisphere much more seriously than the other.

The hemisphere thus involved is the one already referred to as rounded in contour, a condition undoubtedly due to shrinking subsequent upon scleroses and degenerative changes within the corresponding horn of grey matter, associated, as was shown, with the morbid state of the anterior nerve-roots. The condition of the cell groupings here was as follows :—

The *postero-lateral group* of cells is most extensively degenerated.
The *central group* (Ross), together with the former, are the groups most generally affected in this region.
The *antero-lateral* and *medio-lateral* cells are also seriously involved, but less frequently so than the former two groups.
The *internal group*, although occasionally found degenerated, is far more frequently quite intact.

In the opposite hemisphere the conditions are very different.

The *antero-lateral*, *medio-lateral* and *postero-lateral* groups are almost invariably represented by numerous healthy well-branched cells.
The *central group* is frequently found to consist of shrunken and atrophied cells.
The *internal group* is occasionally found degenerated.

The more frequent condition in this region was a perfect development of all the lateral and inner cell-groupings, with perhaps slight disease of the central cells in one hemisphere as contrasted with an extensive implication of all the lateral groups, *especially the postero-lateral and often complete destruction of the central group* in the opposite hemisphere. Even in the healthier hemisphere, however, the marginal cells of the antero-lateral series are often swollen, globose and succumbing to a diseased process;

whilst in one preparation the postero-lateral group was represented by one or two stunted cells, and all the cells of the median group were absent. In *both hemispheres* the central group was frequently represented by mere shrivelled and formless relics of cells situated in the midst of deep stained sclerosed tissue, often with a large dilated and diseased vessel in this neighbourhood. In like manner the morbid process in the postero-lateral group was *always in advance* of the degenerative changes seen in the remaining cell-groups.

The features claiming chief attention, therefore, in the cervical region were as follows.

a. Atrophic shrinking of the cell-groups, chiefly of one hemisphere.
b. This atrophy most apparent in the central and lateral groups.
c. Morbid changes apparent in the anterior roots of this hemisphere.
d. Slight sclerosic shrinking of the corresponding anterior horn, causing diminution in bulk of the whole segment.
e. Limitation of such changes to the lower cervical regions, the cell-groups being more and more healthy higher up, until on a level with the third cervical nerve they are perfectly intact.

The Dorsal Region.

a. Posterior Columns.—As before mentioned, these columns were extensively diseased and consistence so fragile that good sections could not be made without extreme difficulty. Its structure was perfectly riddled throughout by numerous cavities—the perivascular tracts being converted into huge channels, here and there still further distended into oval or spherical dilatations arranged in moniliform series along their course. The membranes of the cord are especially thickened in this region, and the diseased condition of the vascular tunics suggests itself strongly as the origin of the further changes induced in the nervous tissues, since the lesion is decidedly more advanced along the *peripheral border* of the columns, and spreads inwards along the direction of the vessels. The columns of Burdach are much implicated on both sides, but least so in the right hemisphere; the posterior commissural zone is sclerosed to a minor extent, whilst the peripheral margin adjacent to the membranes (sub-meningeal zone) is converted into a zone of pure punctated tissue. In all cases alike it may be stated that the deepest and most advanced morbid change has taken place in this submeningeal zone; still further inwards, acute changes are

seen in the presence of numerous swollen nerve-fibres, colloid bodies, and the extension of sclerosis along the vascular tracts; whilst in the post-commissural zone the vessels of this region have apparently set up a distinct focus of disease, the areas here being mapped out by numerous deep-stained patchy sclerosic islets and largely dilated nutrient branches.

The sclerosed tracts invariably followed closely the course of the diseased blood-vessels.—The columns of Goll near their meningeal extremity, have often large circular cavities excavating their structure, identical to those above described, and the nerve-tissue *immediately* surrounding such spaces was condensed, sclerosed, and deeply stained.

The posterior nerve-roots on either side are extensively implicated, being so greatly wasted that scarcely a solitary axis-cylinder is seen.

β. Lateral Columns.—The *direct cerebellar tract* is involved throughout nearly its whole extent, deeply stained punctated tissue occupying the interspace betwixt the diseased nerve-tubes spreading inwards from the meninges. Its distribution is symmetrical on both sides, but the sclerosic change has upon the left side invaded the *crossed pyramidal tract*, which here contains notably enlarged coarse vessels, similarly affected to those of the posterior columns. The lesion in the cerebellar tract is very distinct; to a certain depth pure fibrillated tissue is found, and deeper still, numerous islets of the same intervene betwixt wasted nerve-fibres and amongst dilated vessels. The implication of the cerebellar tract, however, is less extensive and intense than in the cervical cord. The crossed pyramidal tract has escaped implication on the right side. The changes in the left crossed pyramidal tract cannot be traced downwards in the lowest segment of the dorsal cord, nor yet are they seen at the commencement of this region.

γ. Anterior Columns and Root Zone.—The columns of Turck (direct pyramidal tract) were quite healthy. The anterior root-zones appeared in every respect free from disease, but the submeningeal zone was occasionally invaded from the membranes to a slight degree, and the tissue here was much degenerated and friable. The condition of most of the sections obtained was unsatisfactory to a determination of the state of the anterior nerve-roots, which must therefore be left undecided.

δ. Anterior Cornua.—A notable sclerosic change had affected the left horn of grey matter in this region of the cord. The grey matter had shrunk, so that its transverse diameter was much reduced, the left cornu being represented by a narrow tract of dense tissue one half the width of the opposite horn; at the same

time, whilst undiminished in length, it was more pointed, having lost the more square form of the corresponding cornu of the other side (Fig. E). Its deeply stained condensed tissue contained the relics of diseased cells. Corresponding to this lateral shrinking of the cornu the whole hemisphere was flattened and narrowed laterally, in marked contrast to the rounded contour of the opposite segment, measuring in diameter 3·5 mm. against 4·5 mm. for the healthier hemisphere.

The various cell-groups, fused together as they naturally are in this region, and difficult to distinguish, are extensively atrophied and often entirely wanting; the remaining ones being either swollen, devoid of branches, and with nucleus indistinguishable, or pale, shrunken, and also without connections. The grey substance of the cornu was much diseased; often the site of enlarged perivascular channels, its texture friable and broken down. At the lower end of the dorsal cord the posterior commissure is completely severed from its right lateral connections by one of these large cyst-like cavities, whilst a similar lesion has divided the anterior commissure from the left segment of the cord (Fig. D). Such lesions appear to indicate extension of the disease to the anterior horns, chiefly along the course of the perivascular tracts.

The Lumbar Region.

a. Posterior Columns.—The whole of this tract is the site of large dilated vessels, the lymphatic sheath implicated in like manner but to a minor degree. As in the dorsal region, the part most seriously involved is the "sub-meningeal zone," where the medullated tubuli have entirely disappeared, and to the depth of ·325 mm. a pure punctated fibrous tissue has replaced the nerve-fibre, and extends inwards along the course of the blood-vessels. To a still further depth of ·4 mm. the more acute stage of degeneration is evidenced in the large swollen contour of numerous nerve-fibres.

Patchy sclerosic areas are found, especially along either side of the posterior median raphe, as far as the posterior commissure. The medullated fibres which have especially escaped implication are those found at the *innermost extremities* of the columns of Burdach. The *internal radicular fibres* are observed in sheaves passing into the posterior cornu, apparently not involved to any very notable extent. The peripheral portion of the whole posterior columns is riddled by fissures and cavities, produced as in other regions of the cord.

β. Lateral Columns.—The invasion of the outer and more posterior tracts of these columns is clearly seen; the deep-stained

sclerosed tissue extends forwards across the posterior nerve-roots, and implicates the *lateral margins* and the *crossed pyramidal tracts.*

Numerous large spider-like connective cells are spread over the field, and much punctated fibrous tissue intervenes between the medullated tubuli, which likewise exhibit the segmentation of their medulla as above described.

The medullated tract bordering the grey matter in these columns is invariably intact, but many of the nerve-fibres which pass from the lateral columns horizontally inwards towards the anterior cornua appear to be degenerated. In these latter fibres the *axis cylinder is often seen enlarged.*

γ. *Anterior Columns and Root Zone.*—The columns of Turck are healthy throughout their whole extent on both sides. A similar statement may be made respecting the anterior root-zone, with this limitation; that the more external bundles of *descending medullated tubuli*—in other words, the peripheral or "sub-meningeal zone," seems implicated! In this region a suspicious amount of punctated tissue is found pervading the nervous tracts, so as to appear like an exaggerated development of the peripheral cortical zone of neuroglia, deeply stained. It does in fact appear as though the greater part of the peripheral border of the lumbar cord has undergone more or less sclerosis in the *immediate neighbourhood of the investing membranes.*

The *anterior nerve-roots* are in all cases perfectly intact, and are given off in dense bundles, traced as clearly defined tubuli quite through the columns of the cord from the anterior horns to their point of exit.

δ. *Anterior Cornua.*—The various groups of cells were well represented and healthy throughout the lumbar enlargement, and in all respects the cornua appeared to have escaped invasion by the lesion seen in other tracts.

The Vesicular Columns of Clarke, &c.—Throughout the whole of the dorsal cord the region of Clarke's column was extensively degenerated. The tissue was deeply stained, friable, readily broken down in these sections, and invariably showed diseased cells. The latter were usually inflated and globose, often in the last stages of pigmentary degeneration, shrunken or wholly destroyed.

In the immediate neighbourhood of these columns the vessels were diseased and extremely coarse; whilst occasionally a huge perivascular cavity occupied the site of one of these columns, similar to those found in the posterior columns.

The central canal was, in all regions alike, completely choked up by proliferated endothelial elements.

Examination of the Pons and Medulla Oblongata.

The degenerative change recognised in the posterior columns of the cord was traced upwards along the funiculi graciles and funiculi cuneati into their respective nuclei, the cells of which nuclei were diseased and shrunken. The inner series of arcuate fibres, passing from these nuclei forwards towards the olivary body, were apparently diseased.

The outer division of the restiform tract (restiform body proper) was perfectly free from disease, with exception of a marginal border representing the direct cerebellar tract, which latter column was notably diseased and distinctly mapped out at lower levels, as a deep-stained sclerosed bundle lying in front of the caput cornu, and at higher levels also where it covered the restiform column and the ascending root of the fifth nerve. The lesion in this cerebellar tract was similar in all respects to what was met with throughout the spinal cord, and both columns were affected to about an equal extent.

The ascending roots of the fifth, although close in the proximity of very advanced degenerative changes, *had wholly escaped implication*, and, traced upwards to the level of the emergent fibres of this nerve through the pons, were found to be perfectly normal.

Throughout the whole region of the medulla occupied by the inferior olivary body no other morbid change was detected than the following—

1. Degeneration of the nuclei of cuneate and slender funiculi.
2. Degeneration of the direct cerebellar fibres over the restiform tract.
3. Sclerosis and absence of cells from the *most external region* of the vagus nucleus on each side ("zellenarmer Theil des Vaguskernes" of Schwalbe).
4. Partial sclerosis of the "solitary funiculus" both in the regions of origin of the vagus and of the glosso-pharyngeal nerves.
5. Extreme varicosity of emergent fibres of glosso-pharyngeal around the solitary funiculus.
6. Extensive atrophy of inner arcuate series of fibres passing from the nuclei of the slender and cuneate columns forwards behind the olivary body.

With exception of the partial affection of the column of origin of the lateral mixed system of nerves (vagus, glossophar. and sp. access.) and implication of the outer region of the posterior vagal nuclei, no other cranial nerve, from the third to the twelfth

inclusive, showed indications of disease either in their nuclei or emergent fasciculi.

Each individual nerve was closely examined and the surrounding areas of motor and tegmental tracts were alike normal. Both inferior olives were healthy in every respect.

Examination of a Portion of Sciatic Nerve.

In the fresh state the nerve was of far less firm consistence than in the normal condition, apparently containing much fatty tissue which gave it the aspect and feel of the degenerated sciatic nerve in certain advanced cases of general paralysis. Upon transverse section, the far greater area was occupied by a yellowish, spongy, adipose tissue, three nerve-bundles being seen separated widely by this intervening tissue. Through the centre of this adipose structure coursed two or three minute fasciculi, subsequently recognised as the remains of degenerated and greatly wasted bundles of nerve-fibres. The nerve hardened by chrome-salts had a greasy, unctuous feel, and a large amount of fatty matter had been set free from it in the chrome reagent. Sections taken from fresh and hardened specimens alike exhibited a great surplus of adipose tissue, the cells swollen with fat globules which likewise infiltrated the *epineurium* in all directions. The latter consisted of wavy fibrous tissue, with numerous fatty and degenerated connective cells; whilst larger and smaller oil-globules seen in osmic-acid preparations disposed along the course of these fibres, surrounding its blood-vessels also, and in some cases, penetrating the sheath of the fasciculi, lie disposed upon the outer aspect of the nerve tubuli betwixt them and the *perineurium*. Longitudinal sections showed the nerve-tubuli in most cases in a state of granular disintegration; the medullated sheath having a coagulated aspect, and containing within it coarse and small granules in great abundance. The axis-cylinders had undergone no change, and there was no strangulation of the inter-annular segments by the swollen nucleus.

The segmental nuclei were in most cases not visible, or very indistinct, and faintly stained by carmine. No varicosities of the sheath with proliferating and granular nuclei were observed, such as occurred all throughout the degenerated tracts of the cord.

At times the medullated investment appeared wholly gone, leaving the axis cylinders naked, but otherwise intact. The *endoneurium* in the less affected fasciculi presented no hyperplasia or notable change. In conclusion, the nerve throughout its course

showed extensive granular disintegration of the medullated sheath of the nerve-fibres, with extreme fatty infiltration of the epineurium, and fatty engorgement and increase in the amount of the natural adipose tissue of the nerve.

W. B. L.

EXAMINATIONS OF THE OPTIC NERVE AND RETINA OF THE LEFT EYE.

BY EDWARD NETTLESHIP, F.R.C.S.

The diameter of the optic nerve itself (i.e. not including the outer sheath) is considerably less than that of a healthy nerve for at least half an inch behind the eye (the longitudinal sections do not extend further back). In a longitudinal (axial) section extending from the disc for half an inch backwards the following appearances are observed:—No nerve-fibres can be made out distinctly in the lamina cribosa, nor for a short distance beyond it, the lamina cribosa itself being much thickened by connective tissue, which has the same appearance as that of the normal lamina. The thickened lamina is convex towards the eye as it usually is in cases of swollen disc, but this change in the present specimen seems accounted for by the increase of tissue, without the separation of its fibres by œdema which is seen when the disc is swollen in neuritis. The nerve-fibre bundles begin to be apparent a little way behind the lamina, and become more and more so towards the brain-end of the specimen; but even at half an inch from the eye the nerve is almost as much shrunken, so that many nerve-bundles must probably be wanting.

In all the transverse sections examined, the fibrous trabeculæ between the nerve-bundles are decidedly thickened; this change, however, is far more marked near to the globe than in sections taken further back. There is a corresponding difference in the size and distinctness of the nerve-bundles, the nerve-tissue being scanty and ill-defined near the eye, whilst in the most proximal section these changes are almost confined to the peripheral bundles, and even then are present in a higher degree in some parts of the periphery than others, and a large number of the more central bundles show little if any change. The small blood-vessels in the thickened trabeculæ are full of blood, and their coats are sometimes thick. The intra-vaginal tissue of the nerve-sheath is considerably thickened in some, but not in all places.

The appearances described seem to point to atrophy having begun at the optic disc, or immediately behind it, and to its having

been accompanied (or caused?) by a considerable increase of the connective tissue of the disc-floor itself. Whether the thickening of the trabeculæ further back is due to a primary hypertrophic change in the connective tissue, or is secondary to a previous atrophic process in the nerve-fibres, cannot be conclusively decided from this specimen, as the staining is very imperfect, and I do not know in what fluid the specimen was hardened. It may be pointed out that the evidence in this specimen of the changes having begun at the disc is in accordance with clinical observation.

In the retina, close to the disc, the nerve-fibre layer is much attenuated and composed of well-defined fine wavy fibres with oval nuclei, representing connective tissue or shrivelled nerve-fibres; no ganglion cells are visible, but the other layers, including the rods and cones, are much better preserved than is usual in post-mortem specimens, and look healthy.

I do not know the state of vision in this eye, but from the absence of visible nerve-fibres in the disc, it is difficult to suppose there could have been any sight.

In regard to the ascending changes, it would be interesting to know how long the eye had been blind, if it was blind.

E. N.

This patient presented the following characteristic symptoms of tabes dorsalis.

Ataxy.

Absence of knee-phenomenon, the vastus internus muscle responding to direct percussion.

Shooting-pains.

Giving way of the legs.

Difficulty of walking in the dark.

Numbness and anæsthesia.

Feeling of trunk constriction.

Diplopia.

Optic nerve atrophy.

Argyll Robertson pupils.

Delay in perception of painful impressions.

Bladder trouble.

Besides these, he had for some time complete loss of power in the iliaco-psoas group of muscles, and towards the close of life obstinate vomiting, with abdominal pain (gastric crises).

I have enumerated the symptoms to show that there can be no question regarding the nature of the case. The symptoms during life, confirmed by the appearances after death, point conclusively to tabes dorsalis. No doubt the case presented certain peculiarities. The short duration of the disease, which, if the patient's account is to be trusted, extended only (from the time of attack to death) over a period of twenty-seven months, is remarkable. The degree in which the blood-vessels of the cord are affected, as described by Dr. Lewis, is also unusual. The only case I remember to have seen comparable with it in this respect was that of a female, who was in the hospital a few years since under my care, with disseminated cerebro-spinal sclerosis of rapid progress. In her case there was granular disease of the kidneys. The blood-vessels of the cord were greatly thickened in their walls, the perivascular spaces very large, and there were extravasations of blood, in connection with the diseased vessels, so marked as to be visible by the naked eye on sectioning the cord.

As regards the appearances in the spinal cord presented under the microscope, I quite agree with the opinion expressed by Dr. Lewis. The lesions point, as he has described, to a peri-arteritis apparently originating in the soft membranes, and spreading inwards along the nutrient supply of the posterior columns. That the disease had not remained strictly confined to these columns but had spread, though in far less degree, to other parts of the cord, is what may often be seen in cases of tabes. The tendency to diffusion of this kind seems indeed to me to constitute a considerable difficulty in the way of unqualified acceptance of the hypothesis that the nervous elements are primarily affected. It offers far less objection, on the other hand, to the hypothesis of such a sequence of events as is already generally accepted in the case of disseminated sclerosis.

I do not propose to advance the opinion that the group of symptoms to which we give the name "tabes dorsalis" is always the result of arterial changes, spreading from the soft membranes to the posterior columns of the cord; but I am inclined to think that it is much more often so produced than is commonly supposed. A certain difficulty here presents itself. Can we allow the probability that

a disease identified by the symptoms of which I have given a category as they were observed in the present case, is sometimes the result of changes in the connective tissue bringing about destruction of nerve tubes, and at other times originates in the nerve tubes and spreads to the connective tissue?

The idea entertained by authorities who are entitled to our greatest respect, Charcot and Vulpian, appears to be that the disease may be imitated, so to speak, in consequence of lesions originating in other conditions invading the anatomical district which is affected in tabes. Charcot, for instance, remarked in 1873, that certain lesions of the spinal cord primarily developed outside the posterior columns, might at a given moment invade these at a variable height, and produce, accidentally, as it were, some symptoms of locomotor ataxy. He went on to say that this was *not truly progressive locomotor ataxy*. Vulpian too[1] says, "This disease is primitive or secondary. In the latter case it is produced in the sequel of another affection or previous lesion of the cord. . . . But in these cases the symptomatic expression of ataxy is manifested but seldom with its complete characters, and in such a way that the diagnosis of this complication can be laid down with any certainty."

These cases of secondary origin are supposed therefore to differ materially from the primary disease. I cannot help thinking, however, as I have before suggested[2] that in enquiring into the history of many cases which, when they come before us, present all the characteristics of typical tabes dorsalis, we frequently hear a description of symptoms, often long past, which can only be referred to the occurrence of spinal meningitis, usually of a slight and strictly localised character. In the present instance, as will have been noticed, there was not a symptom wanting of the typical form of the disease, and the chief and perhaps sole peculiarity of the case was the rapidity of its progress to a fatal termination.

In connection with this point I would refer especially to the atrophy of the optic nerve. It is generally agreed that we

[1] 'Maladies du Système Nerveux. Maladies de la Moëlle.' Paris: 1879.

[2] Loc. cit., p. 204.

know of no anatomical association which will explain this lesion as the result of a continuity of nerve elements. That it is not produced (as the sclerosis in the cervical region, for instance, is supposed to be produced) by extension of the irritative process along the nerve-fibrils is evident, both from the fact that it often precedes the spinal symptoms, and also because, as Mr. Nettleship has shown in the present instance, the disease affects the *periphery* of the nerve, and gradually ceases as the centre is approached. So that in the so-called secondary form this interesting peculiarity as regards the position of the degenerative changes in the optic nerve (their centripetal character) is exactly reproduced. Then again there is the transitory diplopia from which, as the history tells us, this man suffered. One cannot help feeling it extremely difficult to explain how it is that these two conditions, peculiar, I think, to this disease, can result from lesions so completely opposed in their origin as those which are thought to characterise primitive and secondary tabes—in the one case an irritative lesion of nerve-fibres leading to hyperplasia of neuroglia—in the other a disease of blood-vessels and neuroglia leading to lesion of nerve-fibres.

Professor Adamkiewicz of Cracow, in a paper read before the International Medical Congress held in London,[1] expresses the opinion that the conception of tabes consisting in a primary degeneration of nerve-fibres in the posterior columns, has been founded on examinations of developed forms of tabes only. He had the opportunity of examining cases in an early phase, and then found the degeneration not diffuse, but involving the connective tissue corresponding to the course of blood-vessels. He made injections of the spinal cord, and ascertained that the signs of degeneration followed the course of the blood-vessels, and involved, 1. The posterior free margin of the posterior columns; 2. The posterior nerve-roots. 3. The septum of the posterior columns. 4. The border between the columns of Burdach and Goll: and lastly, 5. The tracts originating in the inner borders of the

[1] "Tabes ist eine interstitielle Degeneration des Bindegewebes in den Hintersträngen," 'Transactions of International Medical Congress.' London, 1881, p. 163.

posterior horns and converging towards the posterior borders of the posterior columns. Upon the evidence that the beginning of tabetic degeneration coincides with the course of the blood-vessels, he founds the opinion that tabes is an interstitial degeneration of connective tissue. For it is the connective tissue, he remarks, not the nerves, which accompanies the blood-vessels. Tabes thus resemble cirrhosis of the liver, which is an interstitial degeneration proceeding from the connective tissue. This view of the interstitial nature of tabes is clinically of importance, as it explains, he says, the optic-nerve atrophy which frequently accompanies tabes. One has only to suppose that along with the blood-vessels in the cord, coincidently or frequently the blood-vessels of the optic nerves are diseased.

This experience, although very interesting in connection with the present case, is of course by no means conclusive. Indeed it will need much evidence to shake the position which Vulpian defends by arguments of singular force and logical accuracy. But the question is evidently still *sub judice*.

It must be remembered that the vascular supply of the optic nerve passes from centre to periphery. Altered states of nutrition dependent upon disease of the vessels would probably therefore, I would submit, be experienced *first at the periphery of the nerve*, and gradually tend thence upwards. It may be conjectured that a similar explanation might possibly account for those peripheral changes in nerves described by Pitres and Vaillard in an important paper lately published.[1]

In the account of the autopsy it will be noted that a number of calcareous bodies was found in connection with the dural layer of the arachnoid. The presence of bodies of this kind has been before remarked in this disease. Vulpian more than once refers to it, and so also does Westphal, but without attempting, I think, to explain their nature.

It occurs to me that they may possibly lend evidence of some importance as to the relative *age* of the meningeal changes. The changes in the cord can hardly, I should think, have preceded these calcareous deposits in the membrane.

We acquire, too, some useful information from the clinical

[1] 'Archives de Neurologie,' No. 17, 1883. (See 'Abstract' in the present Number of 'BRAIN.')

history as regards the probable time of invasion of the medulla oblongata by the disease. The patient did not suffer from any gastric crisis until about three weeks before his death.[1] The correctness of the opinion advanced by me in 1880, that gastric crises are due to irritation of the nucleus of the vagus caused by extension of the sclerosis into the bulb, has been, I think, practically confirmed, especially by the observations of Pierret and Demange. In the present case Dr. Bevan Lewis found changes in connection with the nuclei of the vagus and spinal accessory nerves, which sufficiently account, it is probable, for the gastric disturbance which was a marked feature in this patient's history during the last three weeks of his life.

[1] 'Lancet,' Feb. 7, 1880.

ON THE RELATION OF THE "AURA" GIDDINESS TO EPILEPTIC SEIZURES.

BY CHARLES E. BEEVOR, M.D. (LOND.), M.R.C.P.

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THE feeling of giddiness occurring as a "warning" or aura before epileptic seizures has been long known, but as far as I have been able to ascertain I have failed to find, in the writings on the subject, an account of any association or relation between the character of the giddiness and the commencement of the ensuing seizure. I shall endeavour in this paper to show that there is some relation between the direction of the apparent movement of the patient and objects surrounding him and the initial rotation of his head at the commencement of the fit.

I think it probable that this apparent movement and the initial rotation are usually in the same direction, or, in other words, that objects appear to move, and the patient feels himself to turn in the *same* direction as his eyes and head are actually drawn in the commencing tonic spasm.

In Vol. III. of 'BRAIN,' p. 192, Dr. Hughlings-Jackson has contributed a valuable paper "On Right or Left-sided Spasm at the Onset of Epileptic Paroxysms," and in speaking of vertigo *in the sense of there being apparent displacement of objects to one side* as occurring in those paroxysms in the first part of which the convulsion is right-sided, Dr. Hughlings-Jackson does not commit himself to any fixed conclusion on the matter. He writes: "I dare not state it as a fact, but my impression is," &c. He dwells on the difficulty of getting trustworthy information from the patient or his friends as to the side on which the initial spasm began or preponderated at the onset of epileptic paroxysms. He mentions various ways

in which the terms subjective and objective are used by medical men, and states how he himself uses them. Admitting and illustrating that the division is arbitrary, he groups vertigo, colours before the eyes, and noises in the ear as crude developments of objective sensations; he groups smell, taste, and the sensation referred to the epigastrium, as crude developments of subjective sensations. Speaking of the so-called "intellectual aura," or, as he prefers to call it, "dreamy state," he says that, if there be also a crude sensation, it is nearly always one of smell, taste, or it is the epigastric sensation. A crude sensation of taste, is very rare, but movements of chewing or tasting are not uncommon with the "dreamy state," and are, he thinks, indirect consequences of discharge of central gustatory elements. With the "dreamy state" there may be vertigo in the sense of the patient himself seeming to turn to one side, or there may be "alteration" in the distance of objects in front of him; there is rarely, he thinks, along with the 'dreamy state" vertigo in the sense of apparent displacement of external objects to one side.

Dr. Mercier has described a case of epilepsy in the last (October) number of 'BRAIN' in which the patient, who had attacks beginning in the left side, experienced the sensation of vertigo in the form of feeling herself turning from left to right, accompanied by noises in the ears. In this case he finds that the vertigo and other sensations felt by the patient were of the objective order, and yet were associated with spasm commencing in the left side, which, as he states, happens to be the wrong side according to the hypothesis that objective sensations are usually associated with attacks beginning on the right side.

I have lately, in the out-patient department of the National Hospital for Paralysed and Epileptic, been investigating the cases of epileptic patients complaining of giddiness before a fit. The word "giddiness" may be taken to mean the apparent movements of objects round a person, or the feeling of the person himself moving round, or these two combined.

The cases, which I found presented the "aura" of giddiness, are seventeen in number. In none of them does the patient state that he feels as though he went the opposite way

to which the objects seem to him to move. In eight cases they complain of feeling themselves and also external objects rotating round from right to left, and nine feel themselves and objects moving in a similar way from left to right.

I have endeavoured to ascertain whether the direction of the apparent rotation of objects and of the patient himself could be found to have any relation to the side on which the initial spasm of the epileptic seizure took place, or, in other words, to find out, in those cases, where the fit begins by giddiness and the feeling of revolution of the patient and objects in one direction—say to the right,—whether the attack begins with actual rotation of the patient's head to the right or to the left side, and whether the spasm begins in the right or left hand.

I think that great stress is to be put on this point (the initial rotation of the head) as a means of determining the starting-point of the fit, for in ordinary bi-lateral epileptic fits it is often quite impossible to find out from the friends, which side of the body is the more convulsed during the clonic stage, whereas the rotation of the head, which is usually present, can be more easily recognised by the friends, especially if they are requested to observe it.

In the above cases in which the vertigo occurred from left to right, in five the fits have commenced on the right side. Of these one patient, who complains of giddiness with apparent rotation of herself and objects towards the right, was an in-patient in the hospital, so that the nature of the attacks was carefully noted, and it was seen that the fit began with turning of the head and eyes towards the right; she was then convulsed on both sides of the body, and I noted immediately after the fit that the right knee-jerk (patellar tendon reflex) was more marked than the left, and that ankle-clonus was obtained better on the right than on the left (see "Knee-jerk after Epilepsy," 'Brain,' April 1882), thus showing that the right side of the body was more affected than the left. In another case with a similar warning of giddiness (this patient after another fit has said that she alone seemed to move to the right, and not objects), the friends state that the head turns to the right, the right arm is drawn up, and the patient is convulsed on the right

side more than on the left. Another patient states that she feels that her mouth is drawn to the right, and that she experiences apparent rotation of herself and objects towards the right; she is also convulsed more on the right side.

I may here say that I have not any record of, what I may call, right-sided vertigo, accompanied by "subjective" sensations, but this is a point on which I cannot speak with any certainty without further data.

In all the cases where giddiness occurred with apparent movement of the patient and his surroundings from right to left, the fits have begun on the left side of the body, and in most cases the head has turned to the left in the initial tonic spasm. One patient volunteers the statement that he feels the left corner of his mouth to twitch, and, according to his friends, at the beginning of the fit his head turns to the left and his left arm is drawn up. Another patient has repeatedly told me that she and the room seem to go round to the left; this is preceded by a sensation of sickness, shooting from the epigastrium to the head. Sometimes, however, she alone feels herself to go round, and not the objects around her, and describes herself to be "so lost" as not to be able to see any objects moving. Her fits begin with rotation of the head to the left. In addition to the left-sided vertigo, another patient feels "funny" and giddy, and as she turns to the left she has the sensation of seeing balls of red, yellow and green in the order mentioned. Her fits commence by the head turning to the left and drawing up of the left arm. This patient, moreover, is very spiteful *before* the fits. Again, a patient had the first fit after a blow on the right occiput; he has now tenderness to percussion on that spot, and sometimes experiences pain there for some days before a fit. He has giddiness with apparent rotation of himself and objects to the left, and his fits begin on the left side. Another patient feels "lost" and faint with the double feeling of left-sided vertigo with actual rotation of head to the left.

Altogether there are twelve patients in which the apparent rotation of themselves and objects is in the same direction as the initial clonic spasm, seven of these being to the left and five to the right. Besides these there are four patients, who

differ from the foregoing in the relation of the direction of the apparent rotation to the initial spasm. These four feel the rotation of themselves and objects to the right; one is described by his friends as being convulsed "all to the left;" another has his head drawn to the left and "struggles round to the left side;" a third case has the sensation of objects and herself turning to the right, and her friends state that the head turns to the left, that the left arm is drawn up more than the right, and the left side is convulsed more than the right. A last case, who is sure that objects and herself seem to go to the right, has left-sided fits, beginning with tingling and drawing up of the left foot, her head turns to the left, and in the fits she is convulsed left more than right. These last two patients are the only ones which entirely oppose the majority of the cases, in that right-sided vertigo is associated with a distinct left-sided spasm, the other three opposing cases not being in any way so certain. I am unable at present to give any explanation why these patients differ from the majority of the cases. One other patient has the sensation of apparent movement of himself and objects from right to left, but I have not yet obtained a definite account of the character of his fits.

In the above cases it is very difficult to be absolutely sure of the facts, when one has to depend so much on the statements of patients and their friends; and as spontaneous statements are worth very much more than answers to questions which are more or less leading, I have avoided as much as possible asking questions which would prompt the patient to give answers in harmony with one's preconceived notions. It is sometimes, however, almost impossible to avoid assisting the patients in their answers; as, for instance, a patient volunteers the information that he feels giddy before a fit, but if he is asked what he means by giddiness, he answers, as though the word giddiness expressed all that could be required or be possible to know, and he is quite unable to explain himself further; on this I usually ask whether he means by giddiness the feeling of faintness or whether he experiences any movement, when perhaps he will jump at the question and say immediately that he swims round or the

room seems to go round; I then point to the window and ask which way it would go in his giddiness, and also make him put himself in the direction in which he would feel himself to be moving.

Sometimes, however, patients, in answer to the question, how they know that a fit is coming on, will say at once, "The room goes round," or "I go round and round," and then the direction can be ascertained without any fear of putting prompting or leading questions.

Notwithstanding the difficulty of proving the correctness of the patient's statements, I think that the large number of seventeen cases, all of which agree in stating, that they and objects appear to move in the same direction—to the right or to the left—must make it highly probable that their statements are true, and that, when patients experience giddiness and the movement of themselves or external objects at the onset of an epileptic fit, they, as a rule, feel to go in a definite direction, and probably objects seem to them to go the same way as they themselves; and further, that in most cases the primary tonic spasm does begin in the corner of the mouth or in the hand and arm on the *same* side towards which the apparent movement of the patient and his surrounding objects takes place. Some of the patients' friends have answered so decidedly my questions, as to which side or part is first affected in the fit, that I cannot but believe that their answers are correct.

I think I ought to mention, that in these cases I have referred to the feeling of giddiness associated with that of movement, either of the patients themselves or of external objects, in a horizontal direction to the right or to the left. I am therefore not referring to those cases, where the patient feels faint or sick, and calls that giddiness, or, on the other hand, to those cases where the patient describes that, as the fit begins, he merely feels his head being drawn round, to one side or the other, but without any giddiness. This feeling is similar to the sensation of the arm and hand being drawn up at the commencement of a fit, and may be unattended with any feeling of giddiness, though of course the two conditions may and do exist together.

In some of my cases the patients express different opinions as to whether the whole of their bodies or only a part seems to move. In some cases of giddiness the head alone feels to turn to one side while the rest of their body does not seem to them to move. I have not as yet been able to get any definite data in these cases, or to find anything to account for the whole body, or the head only, being affected with the apparent movement.

I should state that none of the patients mentioned above have complained of deafness or noises in the ears accompanying the giddiness, and therefore do not come under the class of cases mentioned by Dr. Ormerod in his paper "On Epilepsy in its Relation to Ear Disease," published in the number of this Journal for April 1883.

In connection with the subject of giddiness and movement of the eyes, I would refer to two cases of auditory vertigo, recorded by Dr. Hughlings-Jackson, one of which was published in the number of this Journal for April 1879. In this case the patient's eyes moved during a paroxysm in jerks towards the right side, and he described external objects as seeming to him to move from left to right—in fact, in the same direction as his eyes jerked. A communication from Prof. Donders was read at a meeting of the Ophthalmological Society in 1881 commenting on this case, but as space will not allow me to enumerate his various arguments, I would refer those who are interested in the subject, to vol. ii. p. 213 of the Ophthalm. Soc. Transact.

The other case, which was read by Dr. Hughlings-Jackson at the same Society, in January 1883 (Ophthalm. Soc. Transact. vol. iii. p. 261), was that of a woman who suffered from disease of the right ear, probably set up by infantile otorrhœa, and in whom pressure on the right tragus caused giddiness and a tendency to fall to the left side, and at the same time the eyes moved slowly to the left and came back with a jerk to the right. It was particularly noted that the patient described the apparent movement of external objects from right to left to occur at the moment that the eyes were seen to move slowly to the left, and not when they jerked quickly back again to the right.

Whatever may be the explanation of these two cases, and what bearing they may have on the subject of this paper, I have mentioned them for their great interest, and with the hope that they might support the cases mentioned in this paper. The difficulty is increased by the fact, that in the man the apparent movements of external objects occurred with the rapid jerks of the eyes, and in the woman they occurred with the slower movements of her eyes and not with the rapid jerks. Now in epilepsy one of the most marked motor symptoms at the beginning of a fit is the conjugate rotation of the eyes and head towards one side, and this side—right or left—is the one on which the patient is subsequently in the clonic stage the more convulsed. It is, therefore, rather remarkable, I think, that so many of the cases in this paper—twelve out of seventeen—have felt themselves and the room to go round in the *same* direction as that in which their eyes and head have been seen to turn as the fit began. In fact, these cases seem to agree with Dr. Hughlings-Jackson's first case, concerning which, to use Dr. Jackson's own words, "some of his friends told him that the eyes 'could not' have been moving in the same direction towards which the objects were displaced, and that he must have observed wrongly." I had hoped that the cases I have seen might help to confirm what Dr. Jackson described in his two patients, or perhaps only in the case of the man.

If the statements made by these patients and their friends are true, that in the majority of the cases the apparent movement of the patient and his surroundings is in the same direction as the initial spasm, it is very difficult to explain why this should be so, and I cannot pretend to be able to give an explanation.

I cannot see why there should be any reason to presuppose that objects *ought* to move in the contrary direction to the patient's head and eyes, or why he ought to feel himself going in the opposite direction in which the room seems to go. Our common ideas of giddiness can be produced by prolonged movements of ourselves or of objects before our eyes, and it seems to me that they are different to the feelings of giddiness before epilepsy which are of central origin.

To take two examples of induced giddiness: If one turn

round on one's heel several times from left to right until giddiness is produced, it is said that the room goes in the opposite way, but in reality, as one turns from left to right, the room seems to rush past one from right to left, and when one stops turning, the room still seems to move in the *same* direction, viz. from right to left, while the experimenter feels himself going in the same direction in which he originally turned himself. The feeling of nausea, I find, is much increased if the eyes be kept looking on the ground, the head being bent down, so that the chin touches the sternum and the vertex looks forwards; and if the person then turn himself from left to right, and then on stopping raise his head to the erect position and look at the opposite wall, the phenomenon quoted by Dr. Bradbury ("On Vertigo or Dizziness") from Purkinje is seen, and the movements of the floor are transferred to the wall, which appears to rotate in a direction opposite to those of the hands of a watch; thus seeming to show that the impression of the movement of the floor is reproduced in the same direction on the wall.

On the other hand there is the phenomenon first noticed by Brewster and Addams, referred to in 'BRAIN,' October 1880 ("Optical Illusions of Motion," by Dr. Silvanus Thompson), that when one looked out of a railway-carriage in motion at the ground for some time, and then closed the eyes, undistinguishable forms and patches of light were seen to go before the eyes in the opposite way to which the ground had appeared to be rushing past the eyes.

Addams noticed that when a waterfall was looked at for some time and the eyes were suddenly turned to rocks close by, these appeared to move upwards in a direction opposed to the waterfall. These two latter experiments have been explained by an "unconscious slipping of the inferior and superior recti muscles," but they are more probably due, as Dr. Silvanus Thompson thinks, to a complementary reaction of movement to the eyes, which are tired by a continual movement in one direction.

In the case of turning oneself round on one's heel, the auditory apparatus cannot be excluded from having a share in the giddiness produced, but whether they could counteract

and modify the ocular vertigo, I am unable to say. Unless we can explain these two different results I do not see how we are to presuppose any definite direction in the apparent movement of the patient or his surrounding objects when he feels giddy before an epileptic seizure.

I shall endeavour to pursue the subject further, as I am sure there is a great deal more to be found out in these complicated phenomena.

NOTE ON A CASE OF ANÆSTHESIA.

BY H. B. DONKIN, M.B. (OXON.), F.R.C.P.,

Physician to Westminster Hospital, and to East London Hospital for Children.

THE greater difficulty of establishing the existence and assessing the value of modifications of sensation than of motion as symptoms of nerve-disturbance is of course obvious. Not unfrequently, however, this point is overlooked in reporting cases, and even in drawing important inferences from alleged accounts of sensory abnormalities. At first sight it seems clear that heightened or lowered sense-functions in human beings, depend very largely for the evidence of their existence on the statement of the patient: or in other words are to a great extent "subjective:" while motor disturbance, whether it be in the direction of spasm or paralysis, is much more open to observation or demonstration, and may generally be regarded, with certain limits and precautions, as an "objective" symptom. We all know that a great power of control over the expression of pain or pleasure is possessed by many persons: and that, within the bounds of obvious nervous health, there is often shown a capacity of bearing with apparent indifference the infliction of what most would undoubtedly regard as pain, not to mention the extreme sensibility of others to stimuli which most would ignore as indifferent. Instances of this are too numerous to necessitate quotation. Notwithstanding these facts, it is not uncommon to find the patient's mere statement of heightened or lost sensation, in certain conditions, or after certain applications or remedies, received even by skilled investigators as of equal symptomatic value with demonstrable alterations of motility. Some of the French writings on the neurology of sensation, especially those which deal with the phenomena of anæsthesia as related to "metallo-" and

called physical. How the tendency towards shamming pure and simple is often allied with Hysteria, and how difficult it is sometimes in a given case to disentangle the passive from the volitional factor in the symptom we are studying, is familiar to all who are conversant with nerve-disorders. It is certainly most incorrect and culpable to put down the bulk of hysterico-nervous symptoms to shamming: many of the most distressing expressions of hysterical disease being as completely beyond the control of the will as the pain felt by most ordinary persons on the extraction of a tooth. But it is a wide jump from this position to the belief that mental influence on the body plays but an insignificant part in the production of abnormal sense-impressions. Many observers have now placed it beyond doubt that, in cases where mere shamming could be with the greatest probability excluded, expectant attention can be substituted to a very great extent for the older and vague belief, of late galvanized into a sort of pseudo-vitality in France, in some special influence of metals and magnets on the sense-functions. The experimental methods of "difference" and "concomitant variations," by the use of widely varying applications, in competent hands, have demonstrated the mental factor in many of these cases, thus adding only fresh instances to the numbers already accumulated in such writings as those of Braid, Carpenter, Hack Tuke and others on the influence of the mind upon the body. Yet, erroneous as it may be to refer such cases as many of those reported by M. Charcot and his school to shamming rather than to expectant attention, it is not uncommon to meet with examples of the great difficulty there is in separating these two elements, or, indeed, in establishing the fact of genuine anæsthesia at all; and, when we think we have established it, of coming to a probable conclusion as to the presence or the proportion of controllable elements in its production. I give the following condensed account of a somewhat remarkable and interesting case in illustration of this difficulty.

Robert Warne, aged nearly 13, was admitted under my care at the East London Children's Hospital on October 18, 1882.

The account of his previous condition, as obtained from the mother, was that he had had measles, scarlet fever, and

"magneto-therapy," are notable instances of apparent neglect of the possible vicious intervention of this fallacy in the experiments which they detail and regard as of so great importance.

At the bottom of this constantly occurring confusion in medical and psychological writings, as, indeed, at the bottom of a vast majority of controversies in general, lies a want of clearness of conception of the thing to be expressed, and still more a want of precision in the terms used to express it. In psychology there is an ever-recurrent danger of using physical and psychical terms interchangeably, and in a slip-shod, and sometimes almost disingenuous manner. It would indeed be well in the interests of true physiological psychology if the term consciousness could be altogether abolished, for thus a large amount of metaphysical haze would be dispelled. But perhaps this desirable expurgation of our terminology cannot be made. The term *sensation* though in its physiological application to the sensory nerves, for instance, it stands, or should stand, for the conductivity of impressions from the periphery to an ascending series of centres, is frequently confused with the results of this nervous action as expressed in the feeling (or the consciousness) of the individual. Centripetal nerve action should be kept in our minds quite apart from "conscious" sensation which is the result of some metabolic activity in the higher centres; and if we must use any derivatives of the familiar words "sense" and "conscious" in this context, let us never lose hold of the fact that unconscious sensibility, or unfelt centripetal nerve action, is one thing, and conscious sensibility, or true "feeling," is another. The occurrence, then, of apparent anæsthesia may be regarded as possibly due to an interference with any part of the centripetal nervous apparatus, from the periphery up to the highest sensorial centres.

It should be the primary object in endeavouring to establish or fix the import of any alleged case of anæsthesia, for instance, to eliminate as much as possible the volitional element. Reported anæsthesia may be due, first, to shamming; secondly, to a preconceived mental impression or expectant attention; thirdly, to a mixture of these two elements; and fourthly, may be quite independent of these agencies, and strictly

hurt much. There was possibly some little sensibility to the faradic current in the thumb—especially, as it seems, at the tip, but this phenomenon was not easy to establish. This possibility rested only on a slight twitch of the thumb, occasionally, when the current was applied. The boy denied feeling it. He had a slight discharge from his right ear, with some pain which soon disappeared. He was out of bed every day after admission, making no complaint.

On December 5th he complained of headache, and did not get up. The temperature remained normal. On the 6th he said he had pains in the lower part of his legs. On the 7th there appeared, on examination, to be perfect anæsthesia of both legs, from the soles of the feet up to a line one and a-half inches above the upper margin of the patella. This limit of anæsthesia was repeatedly verified afterwards, the boy being carefully blindfolded, and was tested by the application of all the methods above mentioned of exciting sensation or of inflicting pain.

The boy could walk well, and could stand in any position with his eyes shut without staggering. He said he could not feel the ground at all when walking or stamping; but when the hand was placed under his foot as he stamped, he said he could feel his thigh going up. His knee-jerk was somewhat excessive; no ankle-clonus. The hearing through the skull was good on both sides. On the right side the hearing by the meatus was impaired. He had been subject to a discharge from this ear, off and on, and the tympanic membrane was ruptured. Sight quite good. Colours and objects were accurately distinguished: and the optic disks were healthy in appearance. As to *smell*, he said, being tested, that "Eau de Cologne" and "Jockey Club" smelt nice in the left nostril, nasty in the right; and he stated that he could *taste* salt, &c., only on the left side of the tongue anteriorly, not at all on the right. Sensation to pricking was good all over the tongue.

This case was informally shown for me, by the kindness of a colleague, at a meeting of the Medical Society on Dec. 18, 1882, in order that others might verify the symptoms, which it was thought might disappear as suddenly as they had appeared. The conditions reported above were then ascertained to be present.

whooping-cough, and had suffered much from worms; no diphtheria, or other complaint. Three years before admission he fell down stairs and hurt his chin, since when, his mother says, he has from time to time complained of his head. A fortnight before admission he said he had a pain in his head, which spread down his back and right side, and he lost power in his right arm for quite five minutes. Five days before admission he is said to have had a fit in the night: his "limbs shook and his skin was discoloured." His right thigh also was said to be swollen, and he did not get up next day.

A few days before admission he suddenly fell down, as he was carrying a pail of water. He has never lost his senses, it appears; though he falls down sometimes and feels very faint.

The family history shows that his father has occasionally "fits," which are apparently epileptic.

On admission, the boy appeared to be quite well. There was no complaint or evidence of any motor disturbance; nor did a careful investigation of his organs and functions show anything abnormal beyond what follows.

The only complaint he makes is that of some pain and tenderness on the outer surface of the right thigh, and complete loss of feeling in his thumb. He says he sometimes sees everything red.

Examination showed apparently complete anæsthesia of the skin of the thumb on both aspects: the area involved being exactly limited by a line passing over the carpo-metacarpal joint. The pain in the thigh was not accounted for by examination, and soon passed off entirely. Over the rest of the body the sensation appeared to be good. The tests repeatedly used to the insensitive thumb, and often when the boy was off his guard, were pressure, pricking, pinching, the approximation of a lighted match to the skin, thrusting a needle under the nail and the use of the faradic current.

Subsequent and frequent examinations up to the end of November showed nothing fresh, except that it was thought that he was somewhat indifferent to ordinarily painful sensations over the body generally; though he felt the slightest touch, he said that pinching and pricking did not

On Jan. 3rd, 1883, the boy, still in hospital, remained the same in every respect: a blister was applied to his right thumb, with no effect on the anæsthesia. He was then sent home.

On a visit to the Hospital on Jan. 28, it was found, by the same tests as before, that he had now loss of sensation on the ulnar side of right hand and little finger. This area of anæsthesia extended half-an-inch on to the back and palmar aspect of the hand.

On Feb. 8 I saw him again. He said that since the 5th he had had giddiness and headache, and felt as if everything were turning round him. On the 2nd, when lifting his arm to comb his hair, he had pain in left shoulder preventing him from doing so. On the 3rd, he says he could not raise his arm. The pain is now less. Anæsthesia the same as before.

In the month of April, 1883, I gained admission for this boy into the London Hospital, in order that his condition might be observed by Dr. Hughlings-Jackson. On the day of admission, as appears from the notes kindly sent us by the clinical clerk in charge of the case, the condition was the same as has been already reported. On the 14th, "the patient stated that the anæsthesia of the little finger had disappeared."

"On the 16th a strong faradic current was tried on legs, one pole being placed on his spine and the other on a portion of the anæsthetic area of one leg. Though he said at first that he did not feel it, it was evident by his expression that he did, and that he was trying to bear it. On slightly increasing the current his fortitude gave way, and he shouted out with the pain; which, however, on being questioned, he referred to the back and hip of the same side."

"On the 17th, he stated, the first thing in the morning, that sensation had returned in his legs; and, being tested with the æsthesiometer, sensation was found to be perfectly normal in the previously anæsthetic areas. The faradic current was then tried on the thumb. At the first application he declared that sensation had suddenly returned to that region. One minute previously, according to his statement when the æsthesiometer was used, the anæsthesia was as complete as on admission. (On testing his sense of smell and taste, soon after admission, they were found to be quite normal.) In speaking of the recovery of sensation in his legs, he said, the next morning, that he was

suddenly awakened in the night with shooting-pains in his legs, and these soon left; a feeling of soreness remaining, and sensation being restored."

The case was said to be regarded from the first by Dr. Jackson as one due to "constructiveness."

The result of this case in perfect recovery, in circumstances such as have been detailed, certainly points with some plausibility to shamming as its cause; or, at least, as an element in its causation; and it might be supposed that the boy preferred residence in the Children's Hospital, where he had boys of his own age to play with, and, making himself useful, was a favourite in the ward, to going to school or being in the London Hospital. Yet, it did not appear in any way from his history that he disliked school or home; he did not seem anxious to stay in hospital, or to be considered as an invalid; and there was no evidence of any moral depravity or of hysterical tendency. The boy appeared in every way well and normal, with the exception of his apparent anæsthesia. And, it must be remembered too, that, with the hypothesis of shamming, we have to take into consideration the undoubted and often established fact of there having been frequently no signs or expression of sensation, on the application of what to most of the physically bravest men would be simply torture, and the long remaining exact limitation of the anæsthetic areas, as established by careful and repeated testing, when the patient was blindfolded, and with many other precautions. The anæsthesia of the legs was especially unvarying the whole time he was under observation at the Children's Hospital. On admission to the London Hospital, observations made to map out the area of anæsthesia of the thumb were said to be incongruous and contradictory, and "certainly to point to constructiveness."

If the case may be regarded as one of malingering, the absence of obvious motive is not so interesting as the complete control shown over the expression of suffering; a fact, if it be a fact, of enormous importance in studying the symptomatology of nervous disease, and in connection with all alleged cases of somnambulism, hypnotism, metallo-therapy, &c.

The theory of *expectant attention* will not satisfactorily cover

the case, as all the symptoms were spontaneously complained of by the patient himself.

The elimination of the volitional element altogether leaves the case as an obscure one of organic or more probably, seeing that it speedily recovered, of functional anæsthesia. The distribution of the anæsthesia is, however, not after the pattern of recognised cases. It may occur to some that the application of the faradic current had some specific agency in restoring the sensation, especially as there was some little doubt as to whether, from the first, he could not feel the current when applied to his thumb.

Much of the interest of the case lies in the incongruity of the apparent physical phenomena with the theory of the moral obliquity of the patient. If this boy was shamming anæsthesia when he preserved a perfectly passive countenance as a needle was thrust under his nail, what test shall we apply for the numerous cases of alleged anæsthesia which we see and hear of in neurological cliniques, especially those of Paris? And, on the other hand, we may almost raise the question, what was the sensory condition of the Christian martyrs at the stake, or of the Spartan boy with the fox concealed in his bosom?

It seems on the whole more reasonable to conclude, in the present state of our knowledge, and keeping in view the salient facts of the case, that there was really a want of *conscious sensibility* in this boy, due either to a torpid condition of the sensitive nervous plexuses in those regions of the periphery where stimulation failed to produce its usual effects, and therefore to suspended centripetal conductivity, or to a disordered condition of the sensorium. The former hypotheses appear most probable, when we consider the distribution of the anæsthesia over both legs, the absence of any further abnormal symptoms referable to the cerebrum and the rapid recovery on the application of a strong faradic current. And though other so-called hysterical phenomena were not apparent, the case may perhaps be referred to the same category as some of the sensory disturbances so often seen in confessedly hysterical subjects.

It may be remarked, before leaving this case, that the boy

complained of feeling pain in his legs both at the onset and before the disappearance of the alleged anæsthesia, and moreover that he appears to have been the probable subject of some form of epilepsy, especially when his father's attacks of "fits" are borne in mind. I cannot attempt a further explanation, but I find it hard to believe that the theory of shamming can explain the case away.

In connection with the subject of modified sensations in relation with mental states, the following case, quoted in Dr. Carpenter's work on Mental Physiology, from a Paper by the late Professor Hughes Bennett on the "Mesmeric Mania of 1881," is of some interest. A butcher was brought into the shop of a druggist, labouring under a terrible accident. The man, in trying to hook up a heavy piece of meat above his head slipped, and the sharp hook penetrated his arm, so that he himself was suspended. On being examined he was pale, almost pulseless, and expressed himself as suffering acute agony. The arm could not be moved without causing excessive pain, and in cutting off the sleeve he frequently cried out; but when the arm was exposed it was found to be quite uninjured, the hook having only touched the sleeve of his coat. "No evidence," says Dr. Carpenter, "could be stronger than that afforded by the almost pulseless condition of the patient, as to the reality of the severe pain which he experienced: and yet the pain entirely arose from his mental conviction that the hook had penetrated the flesh of his arm." This case may be compared with the alleged "sensitiveness," to what was called "odylic force," reported long ago by Reichenbach in connection with his experiments with magnets on human beings.

Clinical Case.

CASE ILLUSTRATING CEREBRAL LOCALISATION.

BY JOSEPH WIGLESWORTH, M.D. (LOND.)

Assistant Medical Officer, Rainhill Asylum.

THE following case, in which permanent partial left hemiplegia, and amnesic aphasia were associated with lesions of the right paracentral lobule and left temporo-sphenoidal lobe respectively, appears to be of sufficient interest to justify its publication; and I am indebted to Dr. Rogers, under whose care the patient was, for permission to publish an account of it.

The patient was a female, aged 71, who died in Rainhill Asylum on the 31st of August last, having been an inmate of that Institution for four and a half years.

The only history obtainable was to the effect that she had had two hemiplegic attacks previous to admission. Her condition was pretty uniform during the whole time she was under observation; but it must be mentioned that the hemiplegia—to be shortly described—though never absolute, was most pronounced on admission, and that subsequently a certain small amount of improvement took place. Her condition as manifested at the commencement of the present year may be summed up as follows:—She was a heavy, big-boned woman, with hypertrophied heart, degenerated arteries, and signs of chronic Bright's disease. She had partial left hemiplegia; the arm was practically useless, though she had some slight amount of power in it; the fingers were bent into the palm, and the arm was kept permanently flexed; it could, however, be straightened: the leg was less paralysed than the arm, and patient could walk without assistance, though she did so with a decided limp, and her gait was manifestly insecure; the left leg was distinctly wasted as compared with the right, measuring round the most prominent part of the calf one and a half inches less than on the right side; knee-jerk distinctly exaggerated on the left side; no ankle clonus; no distinct facial paralysis. In addition to this hemiplegia, the patient had great disorder of speech. Her power of articula-

tion appeared unimpaired, and she could articulate some words correctly; but when she attempted to express her ideas, her speech became an unintelligible jargon, in which, though individual words could be deciphered here and there, the incoherent jumbling together of single syllables seemed to form the most prominent feature; when talking rapidly her speech was absolutely undecipherable, not one word she said could be made out; she not unfrequently uttered this gibberish with great volubility. Her mental faculties appeared to be gravely impaired, though it is possible that, owing to the disorder of speech, she got credit for less intelligence than she actually possessed. As a rule, she sat very quiet, seldom attempting to speak unless addressed, and was in general tractable enough; if, however, her usual habits were in any respect interfered with—if, for instance, she was put to sleep in a strange room or bed—she got very restless and excited, and chattered away very volubly, though, as above stated, her speech was quite unintelligible. She clearly understood some things that were said to her; but the impression conveyed was, that her mental faculties were much impaired, that she was, in fact, considerably demented.

On the 25th of August last she was observed to be not looking well, and on examination of chest well-marked pericarditis was found to be present, and she had one-third albumen in her urine; her breathing became embarrassed, and she appeared to be suffering, but made no attempt to complain of anything; she gradually sank and died, as before mentioned, on the 31st of August.

The autopsy was performed on the same day, four hours after death, and the following is an abbreviated account of it.

Cranium.—Skull-cap a little thick; dura mater very adherent to it; three or four ounces of fluid in subdural space. Arachnoid and pia mater a little thickened, everywhere stripped very readily; considerable excess of subarachnoid fluid. Gyri generally somewhat wasted.

Right Hemisphere.—The gyrus fornicatus for the posterior two-thirds of its length had completely disappeared, as also the lower half of the paracentral lobule; these parts had evidently softened down and been absorbed, their position being occupied by a thin membranous bag containing fluid—they had, in fact, been converted into a cyst; the surrounding brain-substance was ochre-stained; the upper half of the paracentral lobule was also superficially softened.

The supra-marginal gyrus was also much atrophied and ochre-stained.

Left Hemisphere.—The middle of the temporo-sphenoidal lobe was occupied by a cyst with ochre-stained margins

similar to the one above described; this involved the upper part of the middle temporo-sphenoidal gyrus, and the middle third of the superior temporo-sphenoidal gyrus; these parts were completely destroyed, and the white matter around, for some distance, was softened.

Cortex of average depth; striæ fairly distinct. Consistence of brain good. *Basal ganglia perfectly normal, as also were the crura cerebri, pons and medulla.*

Brain (immediately after removal) weighed 1064 grammes.

Right Hemisphere	396 grammes	stripped of membranes.
Left	417 „	
Cerebellum	127 „	
Pons	14·5 „	
Medulla Oblongata	6 „	
	960·5	

The main cerebral vessels to their third and fourth ramifications were in an exceedingly atheromatous condition, appearing in some places almost occluded by the thickening of their walls.

As regards the thoracic and abdominal viscera, it may be briefly stated that the lungs were emphysematous; the pericardium acutely inflamed with much effusion of lymph; the heart, weighing 18½ ounces, generally hypertrophied, especially the left ventricle; and that the kidneys each weighed 6½ ounces, had granular surfaces and mottled cortices.

Remarks.—This case is remarkable in the fact that permanent left hemiplegia—incomplete, it is true—was associated with a lesion in the right hemisphere, confined, as regards the motor area, to the paracentral lobule.

Paralysis, partial or complete, of the leg only, in connection with an affection of this lobule, would indeed be quite in accordance with pathological teaching; but the simultaneous implication of the arm is not altogether easy to understand, both central convolutions being intact, as regards their external aspects. The hemiplegia resembled the ordinary kind in the fact of the arm being more paralysed than the leg, nor was there anything—except it were the absence of facial paralysis—to suggest an origin for the affection other than the usual one of a lesion of the pyramidal tract.

The affection of speech would clearly come under the head of "amnesic aphasia," and the localisation of the lesion in the left temporo-sphenoidal lobe seems fairly to agree with that of other recorded cases.

The destruction of brain-tissue was doubtless in all parts due to thrombosis of the vessels supplying the areas in question.

Critical Digest.

RECENT VIEWS ON THE INNERVATION OF THE HEART.[1]

BY BYROM BRAMWELL, M.D., F.R.C.P. (EDIN.)

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Amongst the vital problems connected with the heart, some of the most prominent and important are: (*a*) The construction and mode of action of the mechanism by which its contractions are produced and regulated (its automatic muscular and *motor* nerve arrangements); (*b*) the sensorium is informed of its condition (its sensory nerve arrangements); (*c*) the nutritive condition of its muscular fibre is maintained and regulated (its trophic nerve supply); (*d*) the construction and nature of the nerve arrangements, by means of which it is brought into relationship with the other parts of the vascular system, and with the other organs of the body.

THE AUTOMATIC MECHANISM OF THE HEART.

The nervous mechanism of the heart is extremely complicated, and the manner in which it acts is far from being perfectly understood. It is a subject, however, of the greatest importance, and we must, therefore, consider it in some detail.

In the *first place*, it is necessary to remember that the contraction of the heart is automatic, *i.e.* it is due to impulses arising within the heart itself, and that, in conditions of health, the action of the two sides of the heart, *i.e.* of the two pumps, is synchronous. Under ordinary circumstances the heart beats in a perfectly rhythmical and regular manner, the number of contractions being, in the adult male, about 72 per minute.

Until quite recently it was supposed that the rhythmical action of the heart was entirely due to the periodical and orderly discharge of motor nerve force in the nerve ganglia, which are scattered through the organ; but recent physiological observations, more especially the brilliant researches of Gaskell, seem to show that the influence of the cardiac ganglia is not indispensable, and that the muscular fibre itself, in some of the lower animals at all events, possesses the power of rhythmical contraction.

[1] Part of the first of a course of lectures on the Diseases of the Heart, delivered in the Edinburgh Extra-Academical School of Medicine.

Gaskell's observations seem to prove conclusively, that, in the tortoise, the automatic action of the heart does not depend upon any special rhythmical nervous apparatus, but that it is due to a property of rhythmical contraction inherent in the muscular tissue itself.

It is perhaps premature to conclude that the automatic contractions of the human heart are produced in exactly the same manner as the automatic contractions of the heart of the tortoise. We may, however, safely say, that in man, the rhythmical action of the heart must be due, either to the periodical and orderly discharge of motor nerve force from the ganglia which it contains, or to a rhythmical property possessed by the muscular tissue independently of any special nervous mechanism; and if we may judge by analogy, the latter view is possibly the correct one. And this we may term the *first step* in the comprehension of the mechanism of these complicated arrangements.

In the *second place*, it would appear that the stimulus to muscular contraction is the presence of blood, or rather the presence of blood under a certain pressure, in the cardiac cavities.[1] If the pressure is too low the stimulus is insufficient, and the muscle does not contract. If, on the other hand, the pressure is too great over-distension and a paralytic condition may result. Indeed the recently published experiments of Sewall and Donaldson seem to prove "that, within its working limits of internal pressure, the heart muscle has a remarkable power of accommodating the intensity of its discharges of energy, to the resistance to be overcome.'[2]

"A fact continually forced upon our attention," say these writers, "was the great dependence of the systole, as to its energy and completeness upon the amount of fluid within the heart. Particularly was this noticed in regard to the thin walled sinus and auricles, when isolated in the manner to be described. Within tolerably low fluid pressures, the sinus and auricles become so distended as to be powerless to contract; as the pressure within them is reduced they contract feebly, and when the quantity of blood flowing into them is only just sufficient to bring about full distension during diastole, they contract powerfully, and empty themselves completely at each vent. As the supply of blood is further lessened by lowering the pressure flask, the contractions apparently again become feebler, and in the sinus hardly visible."[3]

"The exact manner in which the cardiac muscle is stimulated

[1] In some of the lower animals, the frog for instance, the heart continues to beat even after the cavities have been cleared of blood, and indeed when they are almost empty of fluid. In the frog, therefore, the presence of blood is not absolutely necessary to produce cardiac contractions, but it is nevertheless probable that the pressure of the blood in the cardiac cavities, under ordinary circumstances, acts as a stimulus, and excites the contraction of the cardiac muscular fibres.

[2] 'The Journal of Physiology,' vol. iii., p. 361.

[3] Loc. cit., p. 361.

by the presence of blood under a certain pressure is obscure. If the muscular contraction can occur independently of the ganglia, we must suppose either that the muscular fibre is *directly* stimulated by the mechanical stretching which it undergoes when the cardiac cavities become distended; or, that the contraction is indirect, and is due to stimulation of the fine nerve fibres[1] which, as Schweigger-Seidel[2] has shown, form a rich plexus in the endo-cardium. If we grant that the ganglia are concerned in the process, we may suppose, as some physiologists have for long held, that the mechanism is a reflex one. A cardiac ganglion-cell may be regarded as the centre of a reflex arc, to which a sensory nerve fibre passes from the endo-cardium, and from which a motor nerve fibre proceeds to the cardiac muscle (see fig. 1); the reflex mechanism being thrown into action by the presence of blood, under a certain pressure, in the cavities of the organ.

Fig. 1.

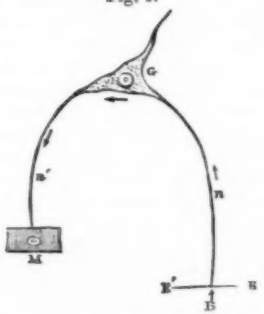


FIG. 1.—*Diagrammatic representation of the reflex mechanism by which (it has been theorised) the cardiac muscle (or muscular coat of the blood-vessels) is thrown into contraction, under the influence of the blood pressure.*

E' E, the endo-cardium; B, the blood in the cardiac cavity; M, the muscular fibre of the heart; G, ganglion cell, the reflex centre; *n*, sensory nerve fibre conducting the impression generated by the blood pressure on the sensory nerve terminations under the endo-cardium to the reflex centre; *n'*, motor nerve fibril conducting the impulse from the reflex centre to the muscle.

In the case of blood-vessels, E E' is the endothelium; the other references will be readily understood.

In the *third place*, the experiments of Gaskell, and of Sewall and Donaldson, seem to show that in the frog and tortoise at least, and when the heart is acting normally, the motor impulse originates in the sinus, and passes from the sinus to the muscular fibres of the auricle, and thence to the ventricle, the rhythm of the sinus determining that of the whole heart.

[1] This view necessarily supposes that these fine nerve fibres are motor.

[2] Quoted by Power, Carpenter's 'Physiology,' p. 276.

Those authorities who believe that the automatic action of the heart is due to the periodical discharge of its motor ganglia, necessarily conclude, in order to explain these results, that the action of the ganglia, which are situated in the sinus venosus, is more powerful than that of the ganglia situated in other parts of the organ; and indeed some authorities have supposed that the ganglia of the sinus venosus are the true automatic centres, while the action of the other ganglia requires to be excited reflexly, or in some other manner. The supporters of the neurotic theory further believe that the sequence of the cardiac contractions (the fact that the contraction of the auricle follows at a regular interval upon the contraction of the sinus, and the contraction of the ventricle at a regular interval upon that of the auricle) is due to nervous influences.

Dr. Gaskell, whose experiments seem to show, as I have already mentioned, that the automatic rhythm originates in the muscular tissue itself, explains the fact that the contractions originate in the sinus, by the peculiar structure of the cardiac muscle in this part of the heart; while he states that the sequence of the contraction of the ventricle to the auricle is not due to separate stimuli passing along nerve fibres from the sinus to the ventricle—not, in short, to any nervous mechanism—but to the fact that a wave of contraction passes directly from the muscular fibres of the auricle to the muscular fibres of the ventricle, through the muscular fibres forming the auriculo-ventricular groove. The pause, or, more correctly, the alteration of rate in the progress of the contraction wave, which takes place between the contraction of the auricle and the contraction of the ventricle, is, he says, due to an alteration in the conducting power, which naturally exists at the auriculo-ventricular ring. "This diminished conducting power or natural block, exists not only because the auriculo-ventricular muscular ring is narrow, and that a somewhat abrupt change occurs in the direction of the muscular fibres along which the contraction wave passes when it reaches and leaves this ring, but essentially because the structure of the muscular fibres here is different from those of the auricle or ventricle.

"The muscle fibres throughout the heart[1] are," he states, "of the same type, any differences which are seen are differences in the prominence of the various structural peculiarities of the cardiac type of muscle, and are not so great as the differences between unstriated and striated muscle fibres. Thus all the muscle fibres are, to a greater or less extent, transversely striated; but the prominence of this striation varies considerably. Similarly the thickness of the fibre, the extent of parallelism of its edges, the size of the nucleus in relation to the size of the fibre, and therefore the extent of crowding of the nuclei in any strip, all present differences in different parts of the heart. The greatest contrast is seen when the muscular fibres of the ground layer in the sinus are teazed out and compared with the muscle fibres of the spongy

[1] He is speaking, it must be remembered, of the adult heart of the tortoise.

tissue of the ventricle. The sinus muscle fibre is thin and delicate, tapering somewhat at both ends, with a large central oval nucleus which causes a distinct bulging of the fibre; the substance of the fibre shows a striation which is decidedly indistinct, presenting often a granular rather than a distinct banded appearance. On the other hand, the ventricular muscle fibre is boldly and strongly striated, it is much thicker than that of the sinus, its edges are parallel, and the thin elongated nucleus is small in comparison to the size of the fibre. The muscle fibres of the reticulated tissue of the auricle are not so large or so coarsely striated as those of the ventricle, though larger and much more distinctly striated than the sinus muscle fibres. Their edges also are more parallel than in the fibres of the sinus. The muscular ring forming the junction of the auricles and ventricle, and to a certain extent the whole junction wall joining the two auricles, are composed of muscle fibres, with a structure intermediate between the sinus and the auricle muscles. The nuclei are large, conspicuous on section both in size and number, the striation is not so well marked as in the bulged portion of the auricles, and the fibres are thin and delicate with somewhat parallel edges.

"Such a structure as above described is very suggestive, not only as an explanation of the causes which occur naturally in the course of the peristaltic wave of contraction, but also of the differences of rhythmical power exhibited by different parts of the heart."[1]

Dr. Gaskell, therefore, conceives "that the variations in the rhythmical power and in conductivity, which are characteristic of the different parts of the adult heart of the tortoise, may all be accounted for on the supposition, that the development of the muscular tissue of the originally tubular heart has not proceeded at the same rate throughout the tube, so that in the adult heart greater variations in rhythmical power are apparent in the different sections of it, than in the original tubular heart; the peristaltic wave of contraction which originally passed smoothly from end to end, passes finally along a tube of irregular calibre, the muscular walls of which have become so modified in their rates of contraction and conduction, as well as in the arrangement of the fibres, as to form out of a simple peristaltically contracting tube such an efficient muscular pump as is represented by the adult heart.[2]

"The conception advanced above, that the rhythmical beating of the heart is due to a series of peristaltic contractions which start from that particular portion of the muscular tissue of the heart in which the property of automatic rhythm has been most largely developed, brings the heart's action into harmony with the rest of the vascular system and with the rhythmical properties which are so often manifested by the less specialized forms of muscular tissue."[3]

Space will not allow me to detail the facts and arguments on

[1] Loc. cit., pp. 72, 73. [2] Loc. cit., p. 77. [3] Loc. cit., p. 80.

which Dr. Gaskell founds this theory, which obviously has most important practical bearings on the pathology of the human heart. I would, however, strongly advise my readers to peruse the original for themselves.

In the *fourth place*, Gaskell has distinctly proved that the ventricle is supplied with afferent nerve fibres, which are "able to regulate the force of the auricular contractions, as well as in all probability the rate of rhythm. It is then conceivable that the function of many of the nerve fibres which pass into the ventricle is by their action upon the force of the auricular contractions to regulate the amount of blood thrown into the ventricle, and therefore the amount of work done by the heart."[1]

But although the movements of the heart are automatic, it is intimately connected both with the sympathetic and cerebro-spinal nervous centres; and its action can, as each one of us so well knows, be readily affected by general nervous influences. But in order that this most important and difficult part of our subject may, if possible, be clearly understood, I must now describe the nervous supply of the heart in some detail.

A description was here given of the nerves supplying the heart, and the subject was illustrated by means of a large coloured drawing, of which the wood-cut on p. 516 is a copy.

The Manner in which the Vagus and Sympathetic Nerves Affect the Heart.

I have now described the construction of the mechanism concerned in the innervation of the heart, and I must next point out the manner in which that mechanism works.

We have already seen that while the movements of the heart are automatic (i.e., are due to impulses arising within the heart itself), they can be profoundly modified by the condition of distant parts; and that the impulses which produce these modifications are conveyed to the heart through certain branches of the pneumogastric and sympathetic nerves. Now, until recently, we might have summed up the effects of these impulses by saying, that *impressions passing to the heart through the pneumonogastric retard, while impressions passing to the heart through the sympathetic accelerate its movements* (hence the terms "*inhibitor*" and "*accelerator*" of the heart which are given to the two nerves respectively); but recent observations seem to show that the action of the vagus on the heart is much more complicated, and that it affects not only the rhythm, but also the force of the cardiac contractions, and that it exerts a trophic influence upon the cardiac muscle. It will be necessary, therefore, to consider each of these functions separately.

The Inhibitory Action of the Vagus.

As the result of the laborious investigations of many able observers, physiologists have concluded:—*Firstly*, that there exists

[1] Loc. cit., p. 92.

in the medulla oblongata a cardio-inhibitory centre, which is continually exerting a restraining influence upon the heart; and *Secondly*, that the action of this cardio-inhibitory centre may be intensified, i.e. the action of the heart may be still further retarded by:—

(*a*) Direct stimulation, i e. by certain changes in the medulla itself.

(*b*) Impressions passing to it from the brain.

(*c*) Impressions passing to it from peripheral parts (reflex stimulation).[1]

"Some authorities also believe that the cardio-inhibitory centre may itself be inhibited, i.e., its restraining power taken off the heart, by impressions passing to the medulla from other parts of the central nervous system, or from the periphery."

The most important local changes which stimulate the cardio-inhibitory centre, appear to be increased blood-pressure within the cranium, and irritative lesions in the medulla or in the neighbourhood of the vagi roots, e.g., inflammatory affections at the base of the brain, etc.; while reflex inhibition of the heart seems to be chiefly brought about by impressions passing to the medulla from the abdominal viscera.[2]

It has also been supposed that the action of the heart may be inhibited by impressions passing from the heart itself. Rutherford's experiments certainly seem to show that reflex inhibition of the heart can be produced in the rabbit by stimulation of the central cut end of the superior cardiac nerve, the impression passing up to the cardio-inhibitory centre in the medulla oblongata, and back, through the vagus and its inferior cardiac branch, to the heart.

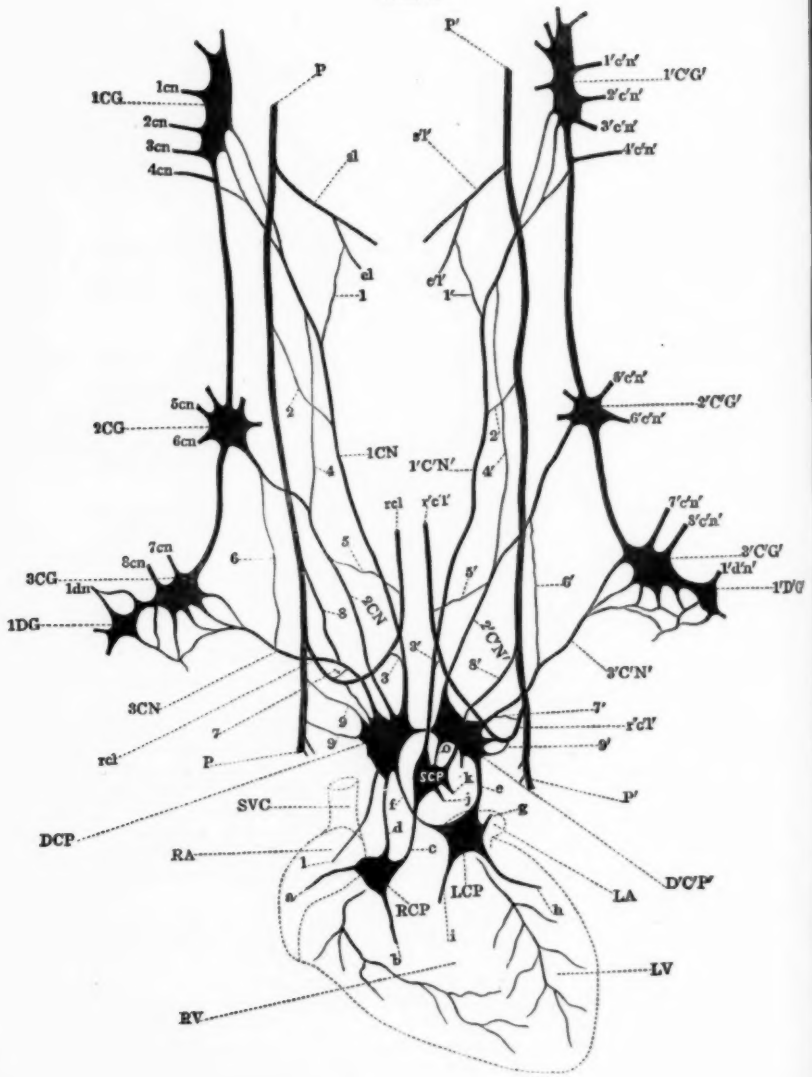
"The *superior cardiac branch* arises by two roots, one connected with its superior laryngeal branch crosses down the neck close to the cervical spmpathetic, and joins one or two branches of the inferior cervical ganglion with which it proceeds to the heart. This branch is a purely sensitive nerve; and is in fact the sensitive nerve of the heart. On cutting it across, and stimulating its inferior (peripheral) end no effect is produced; while stimulation

[p. 518]

[1] Whether the action of the cardio-inhibitory centre is automatic or not, is still undecided.

[2] Powerful stimulation of any afferent or sensory nerve can probably produce reflex inhibition of the heart. Rutherford found that the cardio-inhibitory fibres of the vagus may be thrown into action by stimulating: (1.) The central end of the vaso-inhibitory or superior cardiac branch of the vagus (depressor nerve); (2.) The central end of the vagus of the opposite side; (3.) Almost any sensory nerve, in the case of warm-blooded animals; (4.) The abdominal viscera of the frog; (5.) The splanchnic and sympathetic.—'Lancet,' Dec. 16, 1871, p. 483. Michael Foster states that "the regulative action of the inhibitory mechanism is brought into more or less close connection with all parts of the body."—'A Text Book of Physiology,' p. 174.

FIG. 2.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DCP
RA
D'C'P'
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RCP
LCP
LV
RV

Description of FIG. 2.—*Semi-diagrammatic representation of the nervous supply of the heart.*

RCP, right, and LCP, left coronary plexuses; SCP, superficial cardiac plexuses; DCP, right, and D′C′P′, left half of the deep cardiac plexus; P, right, and P′, left pneumogastric nerves; 1CG, right, and 1′C′G′, left superior ganglia of the sympathetic; 2CG, right, and 2′C′G′, left middle ganglia of the sympathetic; 3CG, right, and 3′C′G′, left inferior ganglia of the sympathetic; RV, right, and LV, left ventricles; RA, right, and LA, left auricles; SVC, superior vena cava.

1cn, 2cn, 3cn, 4cn, 5cn, 6cn, 7cn, 8cn, branches of the right, first, second, third, fourth, fifth, sixth, seventh, and eighth cervical nerves, proceeding to the sympathetic ganglia.

1′c′n′, 2′c′n′, 3′c′n′, 4′c′n′, 5′c′n′, 6′c′n′, 7′c′n′, 8′c′n′, branches of the left, first, second, third, fourth, fifth, sixth, seventh, and eighth cervical nerves, proceeding to the sympathetic ganglia.

1DG, right, and 1′D′G′, left first dorsal ganglia of the sympathetic; 1dn, branches of the right, and 1′d′n′, left first dorsal nerves proceeding to the first dorsal ganglia of the sympathetic.

SL, right, and S′L′, left superior laryngeal branches of the pneumogastric; EL, right, and E′L′, left external laryngeal nerves; rcl, right, and r′c′l′, left recurrent laryngeal nerves at lower part of neck, and in the thorax respectively.

a, branch from right coronary plexus to right auricle; b, branch from right coronary plexus to right ventricle; c, branch from the superficial cardiac plexus to the right coronary plexus; d, branch from the right half of the deep cardiac plexus to the right coronary plexus; e, branch from the left half of the deep cardiac plexus to the left coronary plexus; f, branch from the right half of the deep cardiac plexus to the left coronary plexus; g, branch from the left coronary plexus to the left auricle; h, branch from the left coronary plexus to the left ventricle; i, branch from the left coronary plexus to the right ventricle.

1CN, right, and 1′C′N′, left first or superficial cardiac nerves.

2CN, right, and 2′C′N′, left middle cardiac nerves.

3CN, right, and 3′C′N′, left inferior cardiac nerves.

1, branch from the right, and 1′, branch from the left external laryngeal nerves to the superior cardiac nerves; 2, branch from the right, and 2′, from the left pneumogastric nerves to the superior cardiac nerves; 3, communicating branch from the right, and 3′, from the left recurrent laryngeal nerves to the first cardiac nerves on the right and left sides respectively; 4, communicating branch between the right, and 4′, communicating branch between the left superior and middle cardiac nerves; 5, communicating branch between the right, and 5′, between the left middle cardiac and recurrent laryngeal nerves respectively; 6, communicating branch between the right, and 6′, communicating branch between the left, middle and inferior cardiac nerves; 7, communicating branch between the right, and 7′, between the left inferior cardiac nerves and the recurrent laryngeal respectively; 8, communicating branch between the right inferior cardiac nerves and the trunk of the pneumogastric, 8′, on the left side this branch of the pneumogastric proceeds directly to the superficial cardiac plexus; 9, 9, branches from the trunk of the right pneumogastric, and 9′, from the left recurrent laryngeal nerve to the right and left halves respectively of the deep cardiac plexus.

of its upper (central) end causes pain, retardation of the heart, and, as I shall afterwards more particularly point out, such a striking diminution of blood-pressure, that the term *depressor* nerve has been given to it.

"In the rabbit the vagus gives two branches to the heart, a superior and an inferior cardiac branch (see Fig. 3).

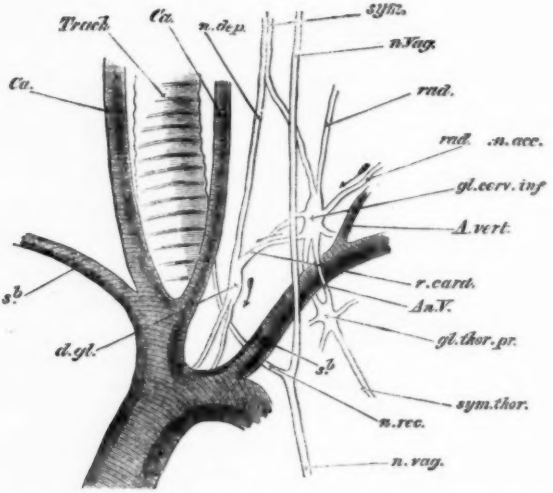

FIG. 3.—*The last cervical and first thoracic ganglia in the rabbit.* (*Left side.*) (*Somewhat diagrammatic, many of the various branches being omitted.*) (*After Foster.*)

Trach., Trachea. *Ca.*, carotid artery. *sb.*, subclavian artery. *n. Vag.*, the vagus trunk. *n. rec.*, the recurrent laryngeal. *sym.*, the cervical sympathetic nerve, ending in the inferior cervical ganglion, *gl. cerv. inf.* Two roots of the ganglion are shown, *rad.*, the lower of the two accompanying the vertebral artery, *A. vert.*, being the one generally possessing accelerator properties. *gl. thor. pr.*, the first thoracic ganglion. Its two branches communicating with the cervical ganglion surround the subclavian artery, forming the annulus of Vieussens. *sym. thor.*, the thoracic sympathetic chain. *n. dep.*, depressor nerve, which, though running by the side of the sympathetic, is really a branch of the vagus, from which it separates higher up. This is joined in its course by a branch from the lower cervical ganglion, there being a small ganglion at their junction, from which proceed nerves to form a plexus over the arch of the aorta. It is this branch from the lower cervical ganglion which possesses accelerator properties—hence the course of the accelerator fibres is indicated in the figure by the arrows.

"The *inferior cardiac branch* leaves the vagus below the origin of the inferior laryngeal nerve, and proceeds directly to the heart.

It is a purely motor nerve, and stimulation of its inferior cut-end gives rise to the same effects as stimulation of the vagus itself (i.e., stimulation above the origin of this branch), viz. (with weak currents) retardation of the heart from prolongation of the diastole, and (with stronger currents) complete arrest during diastole."

In the human subject, two branches of the vagus seem to correspond to the superior cardiac branch of the vagus in the rabbit; while the inferior cardiac branch of the vagus of the rabbit seems to be (mainly at all events) represented in man by the large branch which leaves the vagus trunk at the lower end of the neck (inferior cervical branch).

Now it has been supposed that these two nerves, together with the cardio-inhibitory centre in the medulla, constitute a reflex arc; and that stimulation of the peripheral terminations of the superior cardiac nerve in the heart may generate an impulse which, being conveyed to the cardio-inhibitory centre, and being reflected back to the heart along the vagus, may retard the action of the heart. Whether the superior cardiac nerve has any special function of this description, other than is possessed by all sensitive nerves, is extremely doubtful; and it seems certain that increased blood-pressure within the heart, as a whole, does not, as was at one time supposed, produce reflex inhibition of the organ.

Physiologists are not yet agreed as to the exact manner in which the vagus causes inhibition of the heart.

Until quite recently it was believed that while weak stimulation of the nerve prolonged the diastole, the duration of the ventricular systole being very little affected, more powerful stimulation caused complete arrest during diastole (complete relaxation), the next systole being as it were indefinitely postponed; and it was generally supposed that the vagus inhibited the contractions of the heart by preventing motor impulses being sent from the automatic ganglia to the muscle, i.e., by holding in, as it were, the motor energy of the ganglia; and this would appear to be the view which Sewall and Donaldson take. They think that their experiments "point pretty clearly to the conclusion, that the inhibitory action of the vagus nerve exerts itself in the same indirect manner (as a motor stimulus does), influencing the ventricle by damping in sinus and auricles the excito-motor discharges arising in these parts of the heart."[1]

Dr. Gaskell takes a different view. He has shown:—*Firstly*, that the vagus is able to modify all the great functional attributes of the cardiac muscle, viz.:—

(*a.*) The rate at which automatic contractions are produced. (The *automatic rhythm.*)

(*b.*) The force with which the contractions, more especially the contractions of the auricle, are carried out. (The *force of contraction.*)

[1] Loc. cit., p. 367. 'Journal of Physiology,' iii.

(*c.*) The facility with which the contractions are conducted by the muscular fibres. (The *power of conduction*).[1]

Secondly, that it sometimes produces depression, at other times exaltation of function.

He believes that the vagus may produce standstill or inhibition of the heart (auricles and ventricles) in the three following ways:—

(1.) By producing cessation of its automatic contractions, i.e. by depressing the rhythmical power of the muscular fibres of the sinus in which the automatic rhythm originates. (This has been established in the frog, tortoise, and snake.)

(2.) By reducing the force of the auricular contractions, so that they become invisible, i.e. by depressing the contraction power of the rapidly contracting reticulated muscular fibres of the auricle. (Frog.) The contractions he states may be so small as only partly to be visible, so that to the naked eye the heart appears to stand still.

(3.) By blocking the contraction wave at the sino-auricular ring, i.e. by depressing the conducting power of the muscular fibres connecting the sinus and auricle. (Snake, tortoise, frog.)

The Trophic Function of the Vagus.

As I have previously remarked, Dr. Gaskell[2] has advanced the important theory that the vagus is the trophic nerve of the heart. "Although," he says, "the initial effect of the vagus is to depress some function, its final and most enduring power is to exalt, intensify, and repair that function. Thus, although it slows rhythm, yet its stimulation makes the rhythmical power last longer than it otherwise would, and makes the heart beat with regularity when it was previously irregular; although it reduces the force of the contractions, yet its ultimate effect is to improve and sustain the contraction force, although it may diminish the conduction power, yet in the end it completely repairs that power. *For these reasons*," he says, "*I look upon the vagus as essentially the trophic nerve of the heart.*"

Since this chapter was written I have learned from Dr. Gaskell that the term "trophic" as it is commonly used and understood does not exactly express the function which he supposes the vagus to have over the cardiac muscle. "I have felt," he says, "all along that the word trophic is somewhat misleading as applied to the results of my experiments on the action of the vagus. I wanted a word to express what appears to me to be the fact, in the cold-blooded animals at all events, that the inhibitory process is not destructive but constructive in its nature, but could not find any good term. I therefore used the term 'trophic,' not

[1] In the tortoise the force of the contractions of the ventricle was uninfluenced by vagus stimulation. In the frog, on the other hand, the vagus influences the force of the ventricular as well as of the auricular contractions.

[2] Loc. cit., p. 104.

perhaps in its usual sense, but rather in a sense similar to that of Heidenhain when he divides the nerves supplying the submaxillary gland into secretory and trophic."

The Mode of Action of the Vagus on the Heart.

The exact manner in which the terminal fibres of the vagus are brought into relation with the cardiac muscle is unknown. The effects produced by vagus stimulation, and by the action of certain poisons, have led some physiologists to believe that the arrangement is an extremely complicated one; and in order to explain these results, they have theorised that there existed an inhibitory mechanism within the heart itself, the action of which is strengthened (brought into play) by impressions carried to it by the terminal branches of the vagus, and which in its turn acts upon, i.e. inhibits the ganglionic apparatus more immediately concerned in the production of the rhythmical movements of the heart, i.e. the automatic nerve mechanism.

"The facts and arguments which they have advanced in support of the view that the vagus does not act directly upon the cardiac muscle, but that it acts through some intermediate nerve apparatus are as follows:—

"1. The latent period, which elapses between electrical stimulation of the vagus and the production of its inhibitory action on the heart is twenty times longer than that which elapses between the electrical stimulation of an ordinary motor nerve, and the contraction of its muscle, viz. $\frac{1}{5}$th and $\frac{1}{100}$th part of a second respectively.

"2. A very much stronger current is required to produce inhibition of the heart through the vagus, than is required to produce spasm of a voluntary muscle, when its motor nerve is stimulated.

"3. If the action of the heart be arrested by stimulation of the vagus, the rhythmical contractions after a time return, although the stimulation of the vagus be continually kept up, a fact which may be explained either by supposing that the inhibitory fibres of the vagus become gradually exhausted by the prolonged stimulation, or that the accumulation of nerve force in the automatic ganglia becomes after a time too great to be held back, and that an explosion and muscular contraction consequently take place.

"4. Continued stimulation of one vagus annuls or prejudices the action of the other.

"5. 'The effects of vagus inhibition are perhaps more marked when the electrodes are placed on the boundary line between the sinus venosus and the auricle, than over the vagus trunk itself.'—*Foster.*

"6. If during the complete relaxation which results from powerful stimulation of the vagus, the muscular tissue be mechanically irritated, it is still found to contract.

"While the existence of an intermediate (intra-cardiac) in-

hibitory mechanism between the terminal fibres of the vagus and the automatic nerve apparatus they think is proved by the following circumstances:—

"1. When the electrodes are placed on the boundary line between the sinus venosus and the auricles, more powerful inhibition is produced than when they are applied to the vagus itself.

"2. After the administration of urari, stimulation of the vagus no longer produces inhibition of the heart, but stimulation in the region of the sinus venosus will still do so.

"3. After the administration of atropia, neither stimulation of the vagus nor of the sinus produces any inhibitory effect. It is therefore concluded that atropia paralyses the intra-cardiac inhibitory apparatus itself.

"4. Muscarin and jaborandi in full doses produce exactly the same effect which is produced by powerful vagus stimulation, viz., complete arrest during diastole, an effect which is not prevented by the previous administration of urari, which, as we have seen paralyses the terminal fibres of the vagus, but does not touch the inter-cardiac inhibitory mechanism; but which is prevented by the previous administration of atropia which paralyses the intra-cardiac inhibitory mechanism. Hence it is concluded that muscarin and jaborandi stimulate the intra-cardiac inhibitory mechanism."

Dr. Gaskell's view as to the mode of action of the vagus is different. He has shown that the action of the vagus on the cardiac muscle is in many respects identical with the action of a weak interrupted current. He believes that the nerve acts directly upon the muscular fibre itself, and he argues that the action of atropin, muscarin, and some of the other cardiac poisons to which I have referred above, can be consistently explained upon this view.[1]

[1] "Both atropin and muscarin," he says, "produce the effects above mentioned, by directly modifying the various functions of the cardiac muscle, and in proportion to the stability of the effect so produced is the possibility of a modification of that effect by the direct stimulation of the muscle or by the action of the cardiac nerves. It is partly because the atropin acts on the muscular tissues in such a way as to keep the various muscular powers fixed in a relatively high state of activity, that the cardiac nerves are unable any longer to depress those powers. It is because atropin is able to restore the rhythmical power of the muscle, that stimulation of the vagus and of the sinus is no longer able to produce inhibition; it is because the atropized muscle contracts with the very first stimulus at its full strength, that neither the cardiac nerves nor the interrupted current is any longer able to diminish these contractions; it is because atropin repairs the conducting power that stimulation of the vagus is no longer able to prevent the contraction wave passing from the sinus into the auricles." Again, "atropin not only prevents the action of the vagus nerves by its direct influence on the cardiac muscle, but also by its action on the intra-cardiac nerves themselves, and perhaps especially on their ganglionic nerve cells. Muscarin, on the other hand, influences the muscular tissues, leaving the nervous action intact, at all events, until a very large dose

"Again," he says, "I have shown clearly that the vagus depresses and exalts all the different functions of all the different muscular tissues of the heart, whether the function in question is rhythm, contraction, conduction, tone, or excitability. Also, I have shown that depression of one function is not necessarily accompanied by a simultaneous depression of another function, and so also with the exaltation of function. Further the exaltation of each function is not necessarily dependent upon a previous depression; in each case the primary effect may, under certain circumstances, be exaltation and not depression. Combining these two facts together, we are driven to accept one of two alternatives, either the vagus contains a multiplicity of fibres, which can be divided into two groups after the fashion of Heidenhain, 1. Depressors (Hemmungs-fasern); 2. Augmentors (Verstärkungs-fasern); and further, each member of each of these groups has again its own special function, so that, for instance, the rhythm-inhibiting nerve fibre is different in kind from the contraction-depressing fibre, and so on; or else, the same trophic nerve fibre produces all the different effects observed, according to the nature of the muscle, which it supplies, and the condition of that muscle at the time.

"In this paper as well as in my previous paper, all the facts have tended strongly to prove that the vagus acts in the same manner upon the rhythm of the heart, and upon the force of its contractions, so that if separate inhibitory and accelerator nerve

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has been given. . . . Atropin then not only influences the muscular tissue, but also the nervous structures of the heart in such a way that neither the direct action of the interrupted current nor stimulation of the vagus nerves are any longer able to produce their usual effects upon the various functions of the cardiac muscles.

"The views put forward in this paper," he says, "necessitate a revision of the opinions at present held in respect to the action of a large number of cardiac poisons. The difference between the action of curare and atropin is one of the main arguments in favour of the existence of an inhibitory centre capable of being paralysed by atropin, but not by curare. Both poisons prevent the inhibitory action of the vagus nerve; but while after curare inhibition can still be caused by the direct stimulation of the venous sinus; after atropin this is no longer possible. The inhibition caused by muscarin can be removed by atropin, but not by curare. These facts do not, in my opinion, in the slightest degree prove the existence of an inhibitory centre; they simply confirm the well-known fact that curare is essentially a poison which affects nervous and not muscular stimulus, and give another proof of the affinity of the cardiac muscle and nerve to the ordinary striated muscle and nerve. Thus curare prevents the inhibitory action of the vagus, because it paralyses some part or other of the nerve endings. It does not prevent the inhibition caused by the direct stimulation of the venous sinus, because, unlike atropin, it does not act upon the muscular tissue of the sinus, and therefore cannot prevent the inhibitory action of the current applied directly to that muscle. For the same reason it is unable to influence the inhibitory action of a muscular poison like muscarin."—'Journal of Physiology,' vol. iv., No. 2, pp. 114-116.

fibres exist for the one, separate depressor and augmentator fibres must exist for the other. If, therefore, it can be shown that such diminution and augmentation of the rhythm of the contractions are due not to different influences reaching the muscle, but to the same influence affecting the muscle when its conditions are different, then it is, to say the least, highly probable that slowing and acceleration are also due, not to different nerve fibres, but to the action of the same nerve fibre under different circumstances. In the frog, as I have previously pointed out, such a complete gradation exists between a primary excessive diminution of the contractions and a primary augmentation in consequence of nerve stimulation, as to render the hypothesis that such curves depend upon the simultaneous stimulation of two antagonistic nerve fibres very improbable. I have now proved in addition, that a weak interrupted current applied to the smallest strip of cardiac muscle produces the same two opposite effects, and that here the depressing effect may be removed and the augmenting alone remain, when the condition of the muscle is altered by the application of atropin. In order, therefore, to still hold to the view of specific nerve fibres acting in opposite directions upon the force of the contractions, it is necessary to assume, not only that those nerve fibres possess opposite qualities up to their very termination in the muscle, but that even when the muscle itself is stimulated, the relative action of these two sets of nerves still holds its ground. In addition, atropin would have to be considered as acting upon these two sets of nerve endings, and not upon the muscle, paralysing the depressor nerve endings before those of the augmentor nerves. The experiments of Luchsinger and Szpilman, which lead them to the conclusion that atropin has a special action upon unstriped muscular fibre, those of Bowditch, already referred to, and my own, all point to the conclusion that atropin affects the cardiac muscle directly in consequence of its affinity to unstriped muscle fibre. The whole evidence goes to show that the vagus is a constructive and not a destructive nerve, that the initial depression of function is not of the nature of exhaustion, but is preliminary to a greater functional activity. The phenomenon presented by the muscular tissue of the frog and tortoise under the influence of an induced current, is of exactly the same nature as that seen in the muscle of the snail's heart (as pointed out by Foster and Dew-Smith) when a constant or weak interrupted current is sent through it. In both cases augmentation of function occurs as well as depression; in the one case the evidence is as strong in favour of special inhibitory and accelerator nerves as in the other. Clearly, in the case of the snail, it is simply impossible, as Foster has said, to explain such results by the presence of accelerator and inhibitory fibres in every strip of muscular tissue, when, as a matter of fact, cardiac nerves of any kind whatever have not been proved to exist. Clearly also, whatever explanation will ultimately be found for the action of the current upon the muscular tissue of the snail's heart, will also explain the same phenomena in the heart of the frog and tortoise. The problem which demands

solution is essentially, Why is the condition of the muscle ultimately improved in every one of its functions by the direct application to it of a continuous stimulus, which is not strong enough to produce motor effects? and why is it that improvement of function is preceded in many cases by a diminution of function? When these two questions have received a satisfactory answer, it will no longer be strange that the vagus produces throughout two opposing effects, and the action of atropin will become clear; then the relationship between trophic and motor action will be understood, and the true functions of the ganglion cells found in connection with nerve fibres will be indicated. At present it can only be said that the vagus is the trophic nerve of the cardiac muscle, its action resembling that of a stimulus too weak to produce motor effects, and therefore it is possible that the function of the ganglion cells, in the course of the nerve, is to convert an otherwise motor into a trophic nerve." [1]

"This argument," Dr. Gaskell writes me, is based upon experiments upon cold blooded animals, how far it applies to the warm blooded I (Dr. Gaskell) do not yet know. Eichhorst and Tander conclude that the vagus in the warm blooded contains trophic fibres."

The action of the sympathetic upon the heart is even less clearly understood than the action of the pneumogastric, but it is believed that there exists in the medulla oblongata (or possibly above it), a centre, stimulation of which produces increased frequency of the heart's beat, and it is supposed that this accelerator, or cardiac-motor centre, as it is sometimes called, may be stimulated, i.e. the frequency of the heart's beat may be increased either by direct or reflex stimuli. The influences which throw this centre into action have not been very accurately ascertained, but it is probable that emotion excites the heart through the agency of the accelerator nerves; while diminished blood pressure within the cranium is supposed to stimulate them.

Impulses generated in this centre, the action of which, be it observed, is supposed to be intermittent (and in this respect to differ from the action of the cardio-inhibitory centre, which is supposed to be constant), appear to pass down the lateral column of the cord to the lower end of the cervical region, whence they pass through the nervi communicantes, to the (?) middle and inferior cervical ganglia of the sympathetic, and thence to the heart.

"According to Prof. Michael Foster the accelerator nerves frequently pass along the nerves accompanying the vertebral

[1] It may appear to some that I have entered into undue physiological detail in dealing with this subject, but it must be remembered that the mode of action of drugs is of the greatest interest in connection with the therapeutics of the heart. Further, I am fully convinced that it is only by attention to these minute physiological details that the physician can hope to make much advance in cardiac pathology, a subject on which our knowledge of the ordinary details of morbid anatomy is already so far advanced.

artery, and reach the heart through the last cervical and first dorsal ganglia."[1]

It is supposed from the long latent period which elapses between stimulation of the accelerator and the production of its specific action on the heart, that its fibres terminate in an intermediate ganglionic apparatus similar to that which I have already described in speaking of the termination of the pneumogastric.

Now, although the pneumogastric acts as inhibitor, and the sympathetic as accelerator, it must not be supposed that their action is directly antagonistic. That such is not the case seems abundantly proved by physiological observation. Physiologists have shown, says Dr. M. Foster, that "if during maximum stimulation of the accelerator nerves, the vagus be stimulated even with minimum currents, inhibition is produced with the same readiness as if the accelerator nerves were not acting. Vagus stimulation does not annul, but appears simply to suspend, during its continuance, the manifestation of the accelerator action."[2]

The Relationship between the Heart and the minute Blood-vessels.

In order to conclude the description of the innervation of the heart, I must shortly direct attention to the intimate relationship which exists between the heart on the one hand and the peripheral blood vessels on the other, and to the beautiful self-adjusting mechanism by which alterations in the one are of necessity followed by corresponding changes in the other.

We have already seen the fundamental importance of looking at the heart both as a mechanical pump and as a vital organ; and another point which I must now insist upon—and it is hardly of less importance whether we are studying the heart from a physiological, a pathological, or a clinical point of view,—is *the necessity of taking an all-round view*, so to speak, *of the circulation*. Indeed it is essential to remember that the heart is only a part of the vascular mechanism, and that the condition of the circulation, and consequently the condition of the heart (for the organ naturally adapts itself to the amount and kind of work which it has to perform) depend in no small degree upon the condition of the peripheral resistance, which is in its turn mainly due to the obstruction which the blood meets with, in passing through the minute arteries.

In conditions of health, the minute arteries are always more or less constricted in consequence of a permanent contraction of their middle or muscular coats, and it is to this 'tonic' contraction that the peripheral resistance is in great part due. [The peripheral resistance depends also upon the facility with which the blood passes through the capillary system of vessels; and

[1] The middle and lower cervical ganglia in man appear to correspond to the lower cervical and first dorsal ganglia in the rabbit, through which accelerator impulses have been proved to pass, as shown in Fig. 2.

[2] 'Text Book of Physiology,' p. 175.

since the blood flow through the capillaries may be influenced by alterations in (*a*) the conditions of the capillary walls, (*b*) the condition of the tissues outside the capillary walls, and (*c*) the composition of the blood itself, all of these factors must be taken into consideration as causes of peripheral resistance, and of variations in the blood pressure. In conditions of disease variations in the blood pressure may result from alterations in the "capillary resistance," but these variations are, in my opinion, never so great, either in conditions of health or disease, as those which are due to alterations in the condition of the muscular coat of the minute arteries.]

The degree of constriction is constantly undergoing variations even in health, and is subject to marked alterations in some diseased conditions; hence the amount of resistance which the heart has to overcome in forcing the blood into the capillaries, is constantly changing; and to meet sudden variations of this description a delicate self-adjusting nervous mechanism is provided. But in order that this part of our subject may be properly understood, I must now describe the mechanism by means of which the arterial "tone" is regulated.

There seems good reason to believe:—

(1) That the tonic constriction of the muscular coats of the minute arteries is (immediately) due to the presence of a peripheral neuro-muscular or (purely) muscular apparatus, which may be thrown into action by local stimuli, the most important of which is the blood pressure.

"The peripheral mechanism concerned in this maintenance of arterial 'tone' probably bears a close resemblance to the peripheral mechanism concerned in the production of the rhythmical movements of the heart.

"Ganglionic masses can be demonstrated in the walls of many of the minute blood-vessels, and it has been by some authorities supposed that the blood pressure produces stimulation of the muscular coats of these vessels in a reflex manner (see Fig. 17); but in many vessels (e.g. those in the skin and muscle) no such ganglionic masses have as yet been demonstrated, and in their case we must conclude that the contraction of their muscular coats is due to direct stimulation of the muscular fibre itself, or of the terminal vaso-motor filaments which are distributed to it.

"The view, which supposes that the muscular fibre is itself stimulated by the blood-pressure, is probably the correct one."

(2) That the action of this peripheral neuro-muscular or purely muscular mechanism is, under ordinary circumstances, maintained and regulated by impulses which are being *constantly* sent to it from centres (vaso-constrictor centres) in the medulla and spinal cord, through the vaso-motor nerves; and that the action of these vaso-constrictor centres may be increased or diminished both by direct stimulation or in a reflex manner. In other words, the calibre of the minute arteries, and therefore the peripheral

resistance, may be increased or diminished by central and peripheral causes (stimuli).

"The vaso-motor (i.e. vaso-constrictor) centre of the medulla, which is probably bilateral, is situated immediately above the calamus scriptorius, and extends for some distance into the pons (to 1 or 2 mm. below the corpora quadrigemina). The conducting fibres proceeding from it are believed to pass through the lateral columns of the spinal cord, and to proceed to the blood-vessels chiefly through the sympathetic system of nerves. The vaso-motor centre of the medulla probably regulates the vascular system of the abdomen, the arteries of the skin and muscles being probably mainly supplied by vaso-constrictor fibres proceeding from the vaso-motor centres in the spinal cord. The exact position of these spinal centres is not ascertained.

"The action of the vaso-motor (i.e. vaso-constrictor) centres, which it will be remembered is supposed to be constant, may probably be intensified by:—

"(*a*) Increased blood pressure within the cranium, and local irritative lesions of the medulla.

"(*b*) Impressions proceeding from the cerebrum.

"(*c*) Impressions proceeding from the periphery. It has been experimentally proved that irritation of any afferent (sensory) nerve, such as the sciatic, will produce constriction of the minute blood vessels (especially those of the abdomen) and increased blood pressure, presumably by reflexly stimulating the vaso-motor centre in the medulla."

(3) That the action of the peripheral neuro-muscular mechanism may be inhibited by nervous impulses passing to it from certain centres in the medulla, and (?) spinal cord, through the vaso-dilator nerves; and that the action of the vaso-dilator centres, which is not constant, may be called into play by central and reflex stimulation.

The calibre of the minute arteries may therefore be increased, and the blood-pressure lowered either by inhibition of the vaso-constrictor, or by stimulation of the vaso-dilator centres in the medulla and (?) spinal cord.[1]

"It is not absolutely certain that a distinct and separate vaso-dilator centre exists; some authorities in fact believe that the same centre may at one time evoke vaso-constrictor and at another vaso-dilator impulses; but it seems tolerably well ascertained that there are distinct fibres for the conduction of these two sets of impressions, and for theoretical and teaching purposes it is convenient to describe and represent a distinct vaso-dilator centre.

[1] It must be remembered that the inhibitory action of the vaso-dilators on a peripheral neuro-muscular or muscular mechanism, is theoretical, and it is not definitely proved. "It is quite possible," as Foster says, "that dilation may be brought about in different ways, in different cases, and so also with constriction." —'A Text Book of Physiology,' p. 211.

"The exact course of the vaso-dilator conductors in the cord is yet undetermined, but it seems probable that they pass down in some part of the lateral columns, and reach the blood vessels chiefly through the cerebro-spinal (and not through the sympathetic) nerves.

"The vaso-dilator centre in the medulla is chiefly, if not exclusively, connected with the vessels of the abdominal viscera; and it is most readily thrown into action by stimuli passing to the medulla through the *depressor* nerve, and by impressions passing from the abdominal viscera through the splanchnics."[1]

Now, as I have previously stated, a very striking diminution of blood pressure results from stimulation of the superior cardiac branch of the vagus or *depressor nerve*, as it has therefore been called. It is obvious, therefore, that there is an important connection between the heart and the vaso-dilator centre in the medulla, by means of which the heart and peripheral blood vessels are brought into such close physiological connection, that changes at one end of the circulation, so to speak, are at once attended by compensatory changes at the other. If, for example, the arterial blood pressure is from any cause suddenly increased, a sudden strain is necessarily thrown upon the left ventricle, and it might easily happen that the resistance in front was too great for the force of the pump. Under such circumstances paralytic distention of the left ventricle and death would follow, if it were not for the fact that the undue distention of the cavity and the effort which the heart is making to overcome the resistance, produce irritation of the terminal fibres of its sensitive nerve (the superior cardiac or depressor nerve), in consequence of which a powerful stimulus is sent to the vaso-dilator centre in the medulla, with the result that a sudden dilatation, of the capacious blood-vessels of the abdomen, occurs, and that there is a rapid fall of the blood pressure. The peripheral resistance is in this manner immediately diminished, and paralytic distention of the left ventricle prevented.

It has likewise been thought that the vaso-dilator and vaso-constrictor centres are also connected with the cardio-inhibiting centre in the medulla; and some physiologists have supposed that reflex inhibition of the heart might be produced by the stimulation of the sensitive nerve of the heart (superior cardiac nerve), which presumably occurs in conditions of over-distention of the organ.

[1] This description of the action of the vaso-motor mechanism is not complete. It must of course be remembered, that local variations in the blood supply are being constantly required by the ever-varying needs of particular times and organs, and that as Prof. M. Foster says, "the great use of the whole vaso-motor system is not to maintain a general arterial tone, but to modify according to the needs of the economy the condition of this or that vascular area" ('A Text Book of Physiology,' p. 213). But in studying cardiac affections the changes in the action of the heart, which are induced by local variations of this description, may, for practical purposes, be safely ignored. Hence I have limited my description to those alterations of the vaso-motor mechanism which are sufficiently general to produce definite changes in the action of the heart.

The recent experiments, however, of Ludwig and Luchsinger, and of Sewall and Donaldson, seem to prove that this is not the case, and that increased blood pressure within the heart, as a whole, generally weakens the inhibitory influence of the vagus. The last two observers, however, conclude "that changes of intra-cardiac pressure, *when experienced by the ventricle alone*" (no italics in the original), "are without effect on the cardio-inhibitory function of the vagus."[1] Possibly, therefore, when the ventricles only are over-distended, some reflex inhibition may occur; but even if it does occur, it certainly must be temporary and slight, for the main effect of stimulation of the sensitive nerve of the heart is reflex stimulation of the vaso-dilator centre, and consequent diminution of blood pressure throughout the body; and diminished blood pressure, as we have previously seen, tends to lessen rather than to increase, the inhibitory action of the vagus.

[1] 'Journal of Physiology,' vol. iii., p. 363.

Reviews and Notices of Books.

Body and Will. By Henry Maudsley, M.D. Kegan Paul & Co., 1883. Demy 8vo., pp. 333.

Of all the faculties, qualities or divisions of mind, none is more obscure in nature, more difficult of investigation, or more imperfectly known than that of Will. The psychology of Cognition has been laboriously studied for ages, and our knowledge of it is the accumulated product of twenty centuries of investigation. If the current doctrine of cognition is not complete, it is at least very extensive, and he who cultivates this portion of the science must go again over ground that has been delved by innumerable predecessors. The psychology of Feeling is a younger and less matured science, but it has been prosecuted with vigour, and has reached a fairly advanced state of development. But our knowledge of Will is as yet rudimentary. It has scarcely yet reached a beginning. It is not yet settled what position should be assigned to Will among the other faculties of mind. It is even doubtful whether it should be looked on as a primitive faculty or no. The subject presents therefore to the original worker a great opportunity, and a task of great difficulty, both of which the author of this book appears in his preface to appreciate. "I am not ignorant," he says, "that those who are adepts in the schools of high philosophy may think the essay to be a weak intrusion into their high domains; for I must confess to being unable to use their language with a satisfactory sense of having clear and definite ideas beneath its terms, to having no proper faith in their methods, and to having failed to gather from their works fruits of any practical use." "I have no choice," he goes on to say, "but to leave the barren heights of speculation for the plains on which men live and move and have their being," and he speaks of "the necessity of taking serious account of facts and thoughts which, though they are not philosophy, may claim not to be ignored by philosophy." It appears therefore that Dr. Maudsley is fully alive to the shortcomings of his predecessors; that he approaches the subject from a new standpoint; that he works at it by new methods; that the aim of his book is to be before all things first intelligible, and then practical, and that theorising and speculation are no parts of his object. If we find upon examination that the book is both intelligible and practical, we may congratulate the author upon being successful as far as his main object is concerned, and

may then go on to examine the validity of his doctrines, and the value of his method. If, on the other hand, we find him in the main either unintelligible or unpractical, we must conclude that he has failed in his primary objects, and we need carry the investigation no further. By this means the judgment arrived at will not depend on the opinion of an individual critic; but each reader of the criticism may form his own views of the merits of the book.

The book is divided into three Parts, of which the first treats of Will in its Metaphysical aspect; the second of Will in its Physiological, Social and Evolutional relations; and the third of its Pathological relations. The first part is mainly occupied with a refutation of the mediæval doctrine of free-will, a controversy that does not call for notice in a journal of neurology; but it contains also a chapter on the Physical Basis of Conscious Identity, which is the first chapter that professedly deals with the relation between body and mind, and which arouses therefore a heightened expectation. Proceeding to the study of the body with frank and open mind, Dr. Maudsley perceives that the essential principle of its being and function is a principle of individuation: that it is in fact unit and individual, an *ego*. This is plain and unmistakable on one point—that the body is an *ego*. A few pages further on the author says that "the *ego* is not a constant but a variable. It represents the aggregate of sensations clearly or obscurely felt at any given moment." Here the *ego* appears to be shifting its ground from the body to the mind. It still has extension, however, and "may it not actually be because the *ego* has extension that it can and does think space in every act of consciousness. . . . ? Another consideration: Those who protest so much that the mind has not extension," &c. The metamorphosis is now complete. The *ego* which a few pages back was body is now mind. On p. 16, however, Dr. Maudsley says that "the *ego* is not any abstract entity, but the concrete individual . . . not any extremely sublimed and pure essence, but a feeling, acting, thinking organism." From this it appears that the *ego* is both body and mind, or at least that it is both the individual and the organism. And since things that are identical with the same thing are identical with one another, the individual and the organism are one and the same. But on p. 76 we read, "Are not in truth the individual's conscious memories of his affections and acts far less complete and actual, than the organisms registered memories of its affections and acts?" This sentence necessarily implies that the individual is not the organism, and the organism is not the individual. Thus Dr. Maudsley commits himself to the opinion that the individual and the organism are identical, and are not identical.

The modern doctrine of the parallelism and separateness of mental and cerebral changes is repeatedly accepted and indorsed by Dr. Maudsley; and he vigorously repudiates the supposition that a state of mind can enter into the circle of physical causation and produce an effect upon the bodily processes. On p. 120, he asks whether freedom of choice has, "as every other mental phenomenon confessedly has, a material equivalent in a particular

brain action?" On p. 123 he says, "it will be wise not to describe the objective aspect of mental events in terms of the subjective, nor the subjective in terms of the objective indifferently, but to keep their respective languages apart." Again, p. 301: "Why not resolve to have a definite mental representation of the two invariably and essentially parallel processes when he [the pure psychologist] has to think of either? It would be an excellent check on vagueness of thought and expression, for it would help him to feel that he has a definite meaning in the abstract and somewhat empty psychological terms which he uses so freely, and to make others feel it, and would perhaps render his use of them a little more deliberate, exact, and sparing." Again, on p. 119 he speaks disapprovingly of "those who admit that physical and mental events go along together as exactly parallel phenomena, and who nevertheless bring the correspondence, so exact and constant up to a certain point, to an abrupt end by the arbitrary intervention of free will:" and he goes on to say (p. 120) "that to acknowledge the intervention of an ultra-physical factor in the chain of physical causation" would be "an implicit avowal that causation does not reign in human events, and that the science of human mind must always be metaphysical nescience." Thus there is no mental change without a physical change accompanying it, and to postulate the intervention of a mental factor as a disturbing influence in bodily processes is the negation of all science. On p. 109, however, Dr. Maudsley says that "the body has the power, in virtue simply of its physiological mechanism, without any help of will, to execute most complex purposive acts." This sentence seems to imply a possibility of will helping to execute acts; and in the next sentence this meaning becomes explicit. "The will has not the power to execute only, but it has the power to prevent execution, to hold impulses in check." The remainder of this paragraph speaks of the influence of the will over other mental states, and it is difficult, therefore, to know whether the words quoted mean a power of the will to act on the body; but in the following pages this uncertainty is removed. There the author mentions how "an act of will inhibits the movements of breathing." He explains that memory intervenes to check or inhibit a hasty movement. He thus acknowledges that intervention of a non-physical factor in the chain of physical causation, which, he says, is an implicit avowal that causation does not reign in human events.

How far Dr. Maudsley's endeavour to be practical has succeeded, is for the reader to judge, bearing in mind that practice cannot be founded upon contradictions, and that the questions hitherto dealt with are the most important and fundamental that are connected with the relation between Body and Mind. Even more earnest than Dr. Maudsley's desire to be practical, however, is his desire to be intelligible. He insists so often and so strongly upon the importance of this quality in psychological discussions, that it may be said to be beyond question the chief aim of his book; and if he succeeds in making himself generally intelligible,

he may still claim to have scored a success. Some of the expressions indicative of Dr. Maudsley's desire for accuracy of thought and distinctness of expression have already been quoted, but the prominent place which he assigns to these desiderata as objects to be striven for, appears more from the frequency even than from the earnestness with which he speaks of them. Again and again at intervals of a few pages he urges throughout the book the importance of having clear ideas, and of expressing them clearly. His preface has been already quoted. On p. 5, he cautions us that "many persons do not thoroughly consider whether they distinctly know their own meaning, but deceive themselves in imagining that they have any distinct meaning at all." On p. 15, he says, "it will be well to examine rigorously," "it appears, when we consider the matter closely." On p. 36 he advises us to "let the inquiry be sincere and searching." On p. 69 he says that "it were much to be wished that the philosophers of the study would consider frankly and loyally." On p. 75 he proceeds to the study of the body "with a frank and open mind." On p. 78 he notices how, "recoiling from the danger of intruding upon sacred ground, and from the hardly less deterrent difficulty of resolutely forming clear and definite ideas and expressing them in exact terms and phrases, men have persistently dealt with words instead of things, and with words as things. On p. 84 he recommends "a frank and searching inquiry." On p. 149 he says that "to realise the full meaning of psychological facts, to get clear and exact notions of them in their mental relations is a difficult business, and one which those who have psychology as the method of introspection seem unable to accomplish." These are but a tithe of the instances in which Dr. Maudsley expresses his resolution to form his judgments with the utmost care, and to express them in the plainest terms. If he has succeeded in doing this, he has achieved the main object of his book. Let us take, therefore, a few sentences from the important parts of the book, and examine them with a frank and open mind, to see if we can gather from them clear and exact notions of mental and bodily relations.

The doctrine of the nature of the *ego* has, on account of its importance, already been quoted, and for the same reason it may now be referred to again. Speaking of the sensations which the *ego* represents, the author says (p. 80), "these sensations themselves representing the sum of multitudes of activities that are going on below the threshold of consciousness, and which, albeit unperceived and unfelt immediately, vibrate subtilely (*sic*) in the most intimate and intricate interactions of organic depths, and in the result affect deeply the *tone* of consciousness." It is not certain from the context whether it is the sensations or the activities that vibrate, but let us follow Dr. Maudsley's directions and try to realise the vibration of a sensation or an activity. Having got a definite notion of that, let us add the quality of subtileness, and imagine them vibrating subtilely. Now go a step further, and imagine this vibration occurring in an inter-

action. Again, imagine an organic depth; and yet again, get a firm grasp of the notion of the interaction of organic depths. Now combine all these notions together, and imagine an activity or a sensation vibrating subtilely in the interactions of organic depths. If the reader finds in this expression exact terms and phrases, and can gather from it clear and definite ideas, Dr. Maudsley has succeeded in his object.

Take another most important extract—a declaration of the nature of Mind. "It is," says Dr. Maudsley (p. 128), "*continuous* and *dependent*; for it is a becoming from the basis of all human past through the means of an essential co-operation of surroundings." Let us consider the matter closely and get a clear idea of the basis of all human past; then imagine a becoming from it; then picture to ourselves a co-operation of surroundings; make this co-operation essential, and finally imagine this becoming as happening through the means of this essential co-operation of surroundings. Let the reader follow Dr. Maudsley's instructions; let him make his inquiry sincere and searching; let him not deceive himself into the belief that he finds in these expressions a meaning, if they have no distinct meaning at all; and if he is successful in finding a clear and definite meaning, Dr. Maudsley has succeeded in the main object of his book.

In matters so abstruse as the nature of mind or the physical basis of conscious identity, it is perhaps impossible for an author to make his meaning quite clear, however great may be his desire to do so; let us therefore take some simpler subject, such as the origin of the social habits of ants, and see how the author expresses his meaning in this case. "We may suppose," he says, "that the societies of ants have survived as lessons of what might have taken place in other animals under more favourable auspices, and that their social union became an actuality of organic development, instead of being arrested as a possibility only, by reason of their burrowing habits in the construction of their habitations, of the smallness of their ingeniously constructed bodies, and of their tenacious industry, of their prolific natures, of their numbers and of the strength of their social union." The sentence is clearly expressed; but if we take it in the ordinary meaning of the words, it will appear that the social union of ants became an actuality of organic development by reason, amongst other things, of the strength of their social union; which can scarcely be the author's meaning.

Where the subject-matter is less abstruse, the author is more easily intelligible. He has a chapter on Evolution, to which he adds another process called Involution, of which latter he says that it is easier to entertain the general notion of a process of involution than to put forth an intelligible exposition of it. But he impresses on his readers that they ought "to recognise, investigate, and describe three processes, namely, (*a*) Involution, (*b*) Evolution, and (*c*) Dissolution; which processes, though three when viewed in relation to individual parts, are not three, but three aspects of one process, when viewed in relation to the embracing whole.

"Two leading facts, then, and for us ultimate facts, which it behoves us to apprehend and fix firmly in mind, are—first, that there has been what we may call a nisus of evolution in nature; and secondly, that progressive transpeciations of matter have been events of it. Continuity of nature, certainly, but as certainly not of kind in nature; for the continuity is of different kinds, therefore in some sort a discontinuity, a new kind springing from the basis of the old kind; not continuity by homogeneous, but by heterogeneous generation." No one, I venture to say, will have the least difficulty in clearly apprehending this doctrine who has thoroughly mastered the Athanasian creed.

How far Dr. Maudsley has achieved his aim of resolutely forming clear and definite ideas, and expressing them in exact terms and phrases; how far he has avoided the danger which he points out of dealing with words instead of things, and with words as things; how far he has a definite meaning in the abstract terms that he uses, and makes others feel it, each reader must decide for himself. The foregoing extracts will give the reader ample opportunity of judging of the style and form of Dr. Maudsley's utterances. We may now briefly consider the value of the doctrines that are so presented. "Having," he says, on p. 105, "now done so much to clear the ground, and to set the problem in its true light," &c. It is natural to turn back from such a sentence and see what it is that Dr. Maudsley has done to clear the ground, and to set the matter in its true light. What he has done is sufficiently remarkable. He has propounded a new theory of Mind. We have already learnt that mind is a becoming from the basis of all human past; but this is not the point of view from which it is regarded in the theory now under consideration. The passage in which the theory is set forth is too important not to be quoted entire. It begins as follows:—"Is it not the fact, indeed, that the undulatory theory of light was first suggested by the undulations of sound? In like manner the gulf between the conception of the movements of the cerebral molecules and the self-consciousness of will-energy may well be due to different ways of acquiring them; molecular motion and will be one and the same event, seen under different aspects, and to be known as such one day from a higher plane of knowledge." There is a scarcity of verbs in this sentence, but the meaning is fairly clear. Will is the subjective aspect of what on the objective aspect is molecular movement. The author goes on to say: "Perhaps when that time comes, the theory of an all-pervading mentiferous ether may help to bridge over the difficulty. For if the object and the brain are alike pervaded by such a hyper-subtile (*sic*) ether; and if the impressions which the particular object makes upon the mind be then a sort of pattern of the mentiferous undulations as they are stirred and conditioned within it by its particular form and properties; and if the mind in turn be the mentiferous undulations as conditioned by the convoluted form and the exceedingly complicated and delicate structure of the brain; then it is plain that we have eluded the impassable difficulty of conceiving the action of mind upon matter

—the material upon the immaterial—which results from the notion of their entirely different natures. Here, in fact, is a theory that gets rid at the same time of the gross materiality of matter and of the intangible spiritualities of mind, and instead of binding them together in an abhorred and unnatural union of opposites, unites them in a happy and congenial marriage in an intermediate region, and, if I may so speak, in an intermediate substance; a substance which, mediator-like, partakes of the nature of both without being exclusively either."

From this it appears that the difficulty of conceiving mind in terms of matter disappears if only we imagine the particles of matter as very small. According to Helmholtz, the smallest particle of matter that can ever be distinctly defined under the most powerful microscope is about 1–80,000th of an inch in diameter. Supposing this particle to be spherical, and to consist of water, it would contain, according to Mr. Sorby, 8,000,000,000 of molecules, each molecule consisting, as we know, of three atoms. Notwithstanding the amazing degree of tenuity that this supposition ascribes to the particles of matter, the division between the operations of mind and the undulations of the molecules of water remains absolute; so absolute that a great thinker has said that "we do not possess the intellectual organ, nor apparently any rudiment of the organ which would enable us to pass by a process of reasoning from the one phenomenon to the other." It is for the reader to judge whether the transition will be rendered more easily conceivable, by imagining a still greater attenuation of the particles of matter.

If the mind be itself made up of mentiferous undulations, can these undulations be stirred within the mind by its particular form and properties? and can the reader conceive, by help of this theory, a substance which partakes of the nature of both mind and matter without being exclusively either? A few analogous but much less difficult cases will prepare his mind for the task, and afford him good practice before attempting the major problem. Let him imagine a substance which is partly a triangle and partly a smell, or one which partakes of the nature of both swiftness and sweetness without being exclusively either. If the reader is able by means of the theory of a mentiferous ether to rise from these comparatively simple and concrete conceptions to the much higher conception of the blending of mind with matter, then Dr. Maudsley has achieved the greatest success in mental philosophy of modern times; if not, this theory of mind is a failure.

It will not be necessary to follow Dr. Maudsley's treatment of the various subjects considered in his book. They are sufficiently numerous, varied, and important, to occupy even a larger volume, including, among many others, the question whether animals have souls, the invention of lucifer-matches, the rise of Christianity, the Exodus, the fall of man, the origin of dermoid cysts, the morality of science, the doctrine of the Atonement, the habits of ants and the possible nature of their religion, the immortality of the soul, the failure of the Christian morality, the belief in God and the

existence of the Devil. It is a book *de omnibus rebus et quibusdam aliis.*

It would be presumptuous in me to offer an opinion upon a book of Dr. Maudsley's, and I have not ventured to make any critical observations. In the foregoing notice the reader will find a sufficient account of the book to enable him to form his own judgment upon it, and he will find in addition certain principles laid down upon which he may, I think, safely and properly rely in estimating its value. The issues are largely questions of fact, and the verdict must be left to the voice of the public.

CHARLES MERCIER.

Les Maladies de la Mémoire. Par TH. RIBOT, Directeur de la Revue Philosophique. Deuxième édition. Paris, 1883. 18mo. pp. 168.

To the obligations that psychologists already owe to him, M. Ribot has added another by the production of this charming work, which is written with all the lucidity characteristic of his nation, and with the candour and modesty, as well as the happy union of precise scientific form, with attractive and interesting matter, which his previous works have led us to expect. The work does not contain any new principle; it is a systematic application of established principles to a class of phenomena to which they have not hitherto been so applied, with the result of organising an additional province of our knowledge. The keynote of the book is that memory is the subjective side of a nervous process, and that disorder of memory is the subjective expression of disorder of this process; and the main object of the author is to determine the nature of the disorder that affects the nervous process in each disorder of the memory. In some cases he succeeds in definitely indicating the nature of this disorder; in other cases he does not; and his great merit is that he never attempts to stretch his principles beyond their legitimate tether, or to deceive either his reader or himself as to the success of his efforts. He does what he can; and when he meets with a case that is inexplicable he candidly says so, and does not attempt to hide his ill success under a cloud of meaningless expressions.

The first third of the book is occupied with the consideration of the normal memory, and here M. Ribot lays down a wide and secure basis for the remainder of his subject. Conscious memory, he says, includes three things: the conservation of certain states of mind, their reproduction, and their localisation in the past. But conscious memory or remembrance is but a part, and a small part, of memory. Every case in which a new state is produced in the organism, and is preserved and reproduced, is a form of memory; and the vast majority of such cases have no conscious accompaniment. While

every conscious memory has a biological substratum—implies a physical change in the organism—there are abundance of changes in every respect similar, save that they are not attended by consciousness. Memory is in fact essentially a biological process, and only accidentally and exceptionally psychological. Every case in which a set of movements is acquired, is a case of organic memory. Walking, writing, handicrafts, games of skill, are acquired in precisely the same way, and their acquisition is subject to the same influences as the words of a play or the lesson of a scholar. The two sets of memories resemble one another in every particular save in the presence of consciousness. Nay, they are not materially different even in this respect; for the first are attended by a vivid consciousness, until they are so completely acquired as to have become automatic; while the second, by constant repetition, may cease to have any accompaniment of consciousness. It is manifest that the two memories are essentially the same, and that the same organic process underlies them both. As to the nature of this process, M. Ribot accepts without modification the prevalent modern doctrine. The performance of an act, and the occurrence of a thought, are alike conditioned by the occurrence of a certain process in the nervous tissue; when a particular process takes place in nervous tissue, it leaves in the tissue a permanent modification of such a nature that, if any part of the process is again set going, there is a tendency for the whole process to be repeated. It is scarcely necessary to say that the doctrine that a single remembrance is imprinted on, or stored up in, a single cell is rejected by M. Ribot; but although he deprives the nerve-cells of this part of their prestige, he still attributes to them, it appears to me, a greater share in the process than can fairly be claimed for them. The nerve-cells are the tyrants of the physiological world. Not only can no bodily process be supposed to go on without them, but it is assumed and taught, not only by M. Ribot, but I think universally, that they and their prolongations are the only active portions of the nerve centres. The cell is the dynamite cartridge, and the rest of the grey matter is only the cotton-wool that it is packed in. M. Ribot admits the cell and its prolongations only to a share in the physiological process of memory. He shows that the number of cells—six hundred millions, according to Meynert—is numerically sufficient. He compares them to the letters of the alphabet, of which only twenty-five can be combined to form millions of words. And he infers that the possible number of combinations of the nerve-cells is great enough to satisfy the demands of the memory of the most learned of men. But of the intercellular substance he says not a word. It is time to renew the rebellion set up by the late G. H. Lewes against this despotism of the cell. What right has it to a position of functional supremacy? I know of none. If cells formed the bulk of the grey matter, as they form the bulk of the liver, and if the intercellular substance were present in insignificant amount, then the position might reasonably be conceded to them; but this is not so. When a section of the grey matter from any region of the nervous system is examined under a

low power, the area occupied by the cells (neglecting those of "granular layers") does not preponderate over the area occupied by the intercellular space; on the contrary, the intercellular substance always preponderates, very often preponderates largely, and often is the only element in the field. If the nerve-cell were a constant constituent of grey matter, it might be conceded that the cells were necessary to the occurrence of a nervous process, although it might well be denied that they took the leading part. But their presence is not constant. Not only are there whole columns of grey matter in the spinal cord of man, with few or no cells in them, but in some fishes there are whole lengths of the cord without any cells at all; and in others the whole cord contains only a single cell here and there. The worship of the cell appears to have had its origin in a false analogy drawn between the structure of the grey matter and the structure of the other solid viscera. In the liver, for instance, and in the kidney, the cells are undoubtedly the active element, and the remaining elements have little other function than that of supporting the cells and binding them together. The superficial structural similarity of the brain to these organs appears to have led to the assumption of a functional similarity. Since in the one case there are cells and intercellular material, of which the former are active and the latter inert, and since in the latter there are cells and intercellular material; therefore in the grey matter also the cells are active and the intercellular material inert. The analogy is fallacious and the conclusion vicious. The intercellular material of the liver and the kidney is known to be of a totally different nature from that of the cells; developed from a different layer of the blastoderm, and possessing no community of constitution or property. A large part of the intercellular material of the grey matter is known to be identical in nature with that of the cell, and the remainder is not known to be different from it; it is developed from the same layer of the blastoderm, and the only valid inference with regard to its properties is that they are similar to those of the cell. The processes of the cell are prolongations of its own substance, and are undistinguishable from it in structure. They ramify into a network of fibres, which occupies a great part of the space between the cells. The substance of these fibres is thus continuous with that of the cells, and if we make any assumption about their properties, we must assume that the properties of the fibres are similar to, if not identical with, those of the cell. The remainder of the intercellular space is occupied by the neuroglia, and nothing could be more significant than the fact that while all observers speak of differences between the neuroglia and the nerve tissue, no absolute distinction has ever yet been drawn between them; and the best observers speak doubtfully and differ among themselves as to which cells and fibres should be ascribed to the neuroglia, and which shall be considered purely nervous. There are many excellent reasons why the intercellular grey matter should be looked on as taking an active part in the nervous process that constitutes the physical basis of memory, but these may be now disregarded. Seeing that the cells form but a small portion

of the nerve centre, seeing that the other and larger portion is in part known to be closely similar in substance to the cells, and in the remaining part not known to be different from them, the onus of proof lies upon those who contend that the nerve-cells with their traceable prolongations are alone directly concerned in the function of the grey matter, and proof of this is impossible. The hypothesis is narrow and unreasonable; it rests upon no sound basis; and presumption is entirely against it. The cell-despot, like many another usurper, reigns neither by inherent right nor by the power of his active supporters, but by virtue of a general indifference, and because no one disputes his title. Let us repudiate this *cytolatry*.

Defects of memory are divided by M. Ribot into those which are limited to one class of acquirements, and those which affect simultaneously all the forms of memory. Of the former class aphasia is the type, and of the latter the loss of memory that occurs in old age is an example. General amnesias are again divided into the temporary, such as those which occasionally follow a blow on the head; the periodical, including cases of so-called "double consciousness;" the progressive, which are the commonest, the least curious, and the most instructive; and the congenital. Of each form a few examples are adduced, which have for the most part already done good service in the literature of the subject, and reasoning upon these, M. Ribot goes on to enunciate two important conclusions. The first of these has reference to the curious phenomenon of double consciousness, for which an intelligible physical basis is suggested; and the second is the Law of Regression. The division of consciousness that is found in cases of periodical amnesia is ascribed to an alternating condition of cœnæsthesis. Although the cases are rare, they are so extraordinary that they have attracted much attention, and their facts are tolerably familiar. After some critical event—usually a prolonged sleep—a person awakes with a complete forgetfulness of her whole past life, perhaps with a great change of character. She lives for some time in this New state, and then after another sleep she wakes with a normal memory of all the experiences of her life before her first sleep, and an oblivion of all that had happened in the New state. Another sleep reverses the condition of things. She now remembers the occurrences of the former New state, but is oblivious of everything else; and the Old state and the New state may alternate an indefinite number of times, the acquisitions of each holding good only for the periods of its own recurrence. To find a physical basis for this alternation, M. Ribot investigates first that of the conscious *ego*, a rock upon which many a psychologist has run hopelessly aground, but which affords M. Ribot opportunity for a display of dexterous navigation. The conscious *ego*, considered as it ex sts at any one moment, is the sum of all the states of consciousness then existing; of these states memories form a large part; and it is the sum of these memories which forms the consciousness of continuous identity. The number of different memories that enter into any state of consciousness is very great, and the group so formed is in a state of continual flux

—some memories continually falling out of consciousness and others rising into prominence. In this scene of perpetual change there is, however, one thing that persists. This is the cœnæsthesis—the obscure consciousness which is the result of all the vital actions, and which constitutes the knowledge of our own bodies. It is to changes in the processes that underlie this part of consciousness that M. Ribot attributes the breaks that sometimes occur in our consciousness of continuous identity; and it is to changes in these processes that he would attribute not only cases of periodical amnesia, but also those cases of insane people, who imagine that their bodies are changed into glass or wood, or that their sex is changed, or that they are dead. As to what the precise bodily change is that underlies the change in the cœnæsthesis, or how it is brought about, M. Ribot wisely refrains from guessing.

The Law of Retrogression we are already familiar with, under the name of the Law of Dissolution. The rule is that those requisitions are first lost which are most recent, least often repeated, feebly associated with others—in a word, which are least securely fixed in the nervous organisation. Those memories endure longest which by early acquisition, by frequent repetition and long duration, have been most completely "organically registered."

The consideration of partial amnesias is prefaced by a useful protest against the method of viewing memory as a single integral faculty. There is in reality no such thing as *the* memory; what we experience are *memories*, and in studying the subject this should be always borne in mind. Virtually the study of partial amnesias is limited to the loss of signs (spoken and written words, interjections and gestures); and this is followed by M. Ribot on familiar lines.

A brief consideration of cases of exaltation of the memory leads to the question—Do we ever forget? and this receives the answer that is probably nearest the truth. "It is possible," says M. Ribot, "that certain modifications of cells [I should prefer to say of nervous tissue], and certain dynamical associations are too unstable to endure. We may say, however, that persistence is, if not the invariable rule, at least the rule; that it holds good in the immense majority of cases."

In his conclusion the author shows that while the conservation or organic registration of a memory is an affair of nutrition of the grey matter, its reproduction depends on the state of the circulation therein.

The account here given will be enough to show that M. Ribot has produced a very valuable work, but it cannot convey the charm of the author's style, nor can it hope to emulate the clearness and precision of language which makes a most complex and difficult subject appear childishly easy. Much, in fact most, of what M. Ribot says, has been said before, but it has never before been said so clearly and unmistakably, nor in so convincing a manner.

CHARLES MERCIER.

Traité des Névroses. Par A. AXENFELD. Deuxième édition, augmentée de 700 pages, par HENRI HUCHARD; pp. 1195. Librairie Germer Baillière et Cie., Paris, 1883.

IN 1863 Axenfeld published the first edition of this work. While preparing the second, he in 1876 succumbed to a mortal illness. Anticipating the fatal termination of his malady he confided the conclusion of his literary undertaking to Dr. Huchard, who, during the year 1883, has produced the volume before us. This practically consists of the combined labours of both authors, as the original work of Axenfeld, consisting of 520 pages, remains essentially as before, but has been augmented to the extent of nearly 700 pages by M. Huchard, whose share can always be recognised by its being placed between parentheses. The volume as a whole, while professing to retain the general plan and originality of Axenfeld, has been brought up to the level of scientific knowledge at the present date, and all the modifications and improvements of the last twenty years have been added by the Editor.

Of an undertaking of this size and importance it is impossible at the present time to give a minute critical digest, it is sufficient to say that the work consists of a series of memoirs on the different neuroses, each of which is in itself complete, and embraces in a brief yet comprehensive manner all that is known of the subject. To every section is attached a most voluminous bibliography, the compilation of which represents an enormous amount of research and labour, and which supplies most valuable and extensive material for reference. The text is clear, concise, and well arranged, and from its completeness and general excellence, the work may be looked upon as a standard one, and well worthy to be placed on the shelves of every physician. Having thus summed up the merits of the volume, it is not necessary to further dilate on its details; but there are some general questions with reference to the subject of which it treats, which may be discussed with interest.

In the first place it may be asked, What is a neurosis? Axenfeld defines the term as follows. "A morbid state, most usually apyrexic, in which there exists an exclusive or at least predominant modification of the intelligence, sensibility or motility, or all these faculties combined; a morbid state in which there is this double peculiarity, that it may be produced in the absence of any appreciable lesion, and does not by itself induce profound or persistent structural changes." Like most other definitions of natural processes, this is not a perfect one, and even its author admits that it is not complete, but only approximative. At the same time, it as nearly as possible expresses the position of a very complicated state, and one very difficult to differentiate in words. The term "neurosis," although of British origin—Cullen having been the first to introduce the word—is less employed in this country than it is abroad, the expression "functional nervous

disease" having been substituted for it. There can be little doubt, however, that the former is the preferable terminology, and Neurosis, a word involving little theory, is a convenient term to express that class of affections, to which the appellation of functional has added confusion and given rise to much controversy.

Next arises the question, Is there a place in medical nosology for these neuroses or so-called functional nervous disorders? In practice, two classes of disease are met with, broadly differentiated by their pathological conditions and their clinical features. The first of these is characterised by the existence of a definite and demonstrable lesion, consisting either of morbid new formation or of tissue degeneration. This not only leads to abnormal signs, as a direct result of the structural alterations, but to a series of symptoms due to perturbation of the functions of innervation. Such signs and symptoms bear a constant relation to the lesion which caused them, and may therefore be looked upon as secondary events, as they disappear when the primary change no longer exists. Disease under such conditions is called organic. To be distinguished from this is the second class of disorder under consideration. Here precisely the same symptoms may ensue, but they arise idiopathically, and are not the result of any pre-existing lesion or demonstrable tissue change, nor are they accompanied by any of the signs resulting therefrom. As in organic disease, the symptoms themselves are only modifications of the functions of the nervous system, but they are independent and possess certain peculiarities. They are induced by causes which do not give rise to structural change; they are characterised by extreme mobility and readiness to modification; the same influences which produced them may effect their sudden and complete recovery without a trace being left of their existence, and however long they persist, they do not necessarily lead to further derangement or danger to health and life. Such a train of conditions has been termed, a neurosis, or functional nerve-disorder.

Between these two forms it must be admitted that there is no fixed line of demarcation, and that the definition of each is purely an arbitrary one; at the same time it may be conceded that for practical purposes, both on pathological and clinical grounds, such a distinction may be permitted. In both, if the symptoms differ in character and behaviour, they are identical in kind, consisting, as they do, of a modification of the functions of the nervous system, of increase, diminution or perversion of the intelligence, motility, sensibility, or sympathetic processes. In organic diseases these are the secondary results of a definite lesion; while in functional disorder they arise independently, constituting, as it were, a simple instability of natural acts.

It has, however, been maintained that the well-marked and often permanent abnormalities that are seen in some neuroses must be represented by structural change. In one sense this cannot be denied. Every healthy act or function we may hypothetically assume to be accompanied by molecular movement in the nervous centres. This, it is obvious, can never be detected or anatomically

displayed. Should such acts or functions become disorderly, as during violent emotion, we may conceive an atomic change to accompany them, but this we cannot hope to demonstrate. Finally, should this irregularity become persistent, it is called disease, the symptoms of which are apparent, but the molecular movements which led to them would for ever remain unknown. Such a theory may explain what is known as functional disease, which may be described as a persistent modification of the condition in health, without further structural alteration than that accompanying functional activity.

Whether prolonged functional disorder ever leads to permanent tissue-disorganisation is not known. Experience tells us this is not necessary, as persons may suffer for years from symptoms of the greatest severity which may rapidly and completely recover. On the other hand, the progress and termination of certain cases of chronic hysteria and other neuroses would suggest that it is not impossible, as their symptoms and termination seem only to be explained on such an hypothesis.

It does not of course follow that because an organic lesson is not demonstrated the disease in consequence is a functional one. There are many disorders in which the present state of science fails to indicate any objective changes, yet which are not placed under this denomination, as their symptoms and termination seem to indicate that although we cannot prove a structural degeneration, in all probability such exists. In this, as in all other difficult circumstances, in deciding to which category any particular malady is to be placed, we must be guided by the general features of the case, and by the application of broad principles.

It would therefore appear that there is room for a special class of disorder, not strictly defined, it is true, to which, for convenience, we give the name of neurosis. It is an affection of universal prevalence, and if not actually endangering health or life, is accompanied with the profoundest suffering and misery. Of the exact nature of the condition we know nothing, for, as already pointed out, not only are the anatomical alterations incapable of demonstration, but from their character it is likely that they will always remain so. The manifestations only are apparent, and although these often are severe and protracted, they happily offer encouraging hopes for medical treatment, as from their *nature* there is nothing to prevent complete recovery.

Although the pathology of the neuroses has not advanced, and still remains exposed to the attacks of hypothesis and speculation, our general information concerning the phenomena presented by this class of diseases has steadily progressed, and a review of the work before us to a certain extent indicates the additions to science which have been made during the last quarter of a century. Twenty years ago, when Axenfeld published the first edition of his book, as M. Huchard points out, an exact classification of the neuroses was impossible, our general knowledge of nervous diseases was confused, their study was the more difficult as most of the affections were ill defined, their symptomatology was vague, their diagnosis

undecided, their etiology obscure, and their treatment too often left to chance or empiricism. Since then our acquaintance with these details has made great strides. In addition to the three classes of neuroses described by Axenfeld, namely those of intelligence, sensibility, and motility, we now recognise those of the vaso-motor system. On some questions our information has undergone little change and the description of tremor, paralysis agitans, the peripheral and visceral paralyses, neurasthenia, &c., remains the same as it did twenty years ago. Other subjects, however, have received considerable development, and among these may be mentioned the neuralgias in general, and in particular, migraine, angina pectoris, vertigo, functional spasms, contracture, epilepsy, chorea, and hysteria. In all of these, if no actual discoveries have been made, our knowledge has been extended, and their nature has become more fully understood. On the important subject of hysteria the Editor has more especially found it necessary to make large additions to the text of twenty years ago, and in the chapter devoted to this subject is embodied the discovery of the various phenomena connected with hystero-epilepsy, hypnotism, and metallotherapy, which of late years has attracted the attention of the profession, especially in France. In addition, our information on this neurosis has been augmented by researches on its visceral symptomatology; the relations between it and other morbid states; its vaso-motor and secretory disorders; its convulsive phenomena, contractures, and paralyses; its intellectual and mental abnormalities, and its treatment. In an affection so universal and always so distressing—although in England we do not see its worst forms—all such improvements in knowledge must be of the greatest practical importance. M. Huchard endeavours more especially to penetrate into the mental and intellectual peculiarities, and to analyse the conditions of the character and mind, in short, to study the patients as well as the disease. The Editor also gives treatment the fullest consideration, and he remarks that many distinguished men occupy themselves with scientific research on disease, but place therapeutics in a secondary position. The patient does not look upon the question in this light, and M. Huchard declares himself in this respect an ally of the sufferer. In this we entirely sympathise with him, and there can be little doubt that of all the ills to which mankind is doomed, the neuroses are those which cause the greatest amount of suffering, and at the same time hold out the most encouraging prospects to treatment. Although many such disorders become chronic and intractable, there are many in which a physical and moral *régime* is attended with the greatest success, and it is probable that as the affection is better understood and more easily recognised, prophylactic and curative measures will diminish a large amount of misery.

The diagnosis of the neuroses is frequently attended with great difficulty, as their origin is obscure, and the symptoms may simulate in every respect those of organic disease, or may be complicated with them. It is clear that the treatment suitable

for the one is positively contra-indicated for the other. Thousands of persons have drifted into a state of chronic invalidism who, had their true condition been recognised, might readily have been cured; and, on the other hand, many have been neglected, or even ill-treated, on the supposition that they were suffering from a trifling or imaginary disorder, when in fact they were the victims of an intractable disease. Such a work as that before us is eminently calculated to dispel the clouds from the horizon of ignorance, entering as it does into the whole subject in a liberal spirit of scientific and practical enquiry.

M. Huchard, while giving credit to the observers of all nations for advancing the science and practice of neurology, claims for France a pre-eminence in that particular, recording in proof the names of a phalanx of distinguished men who will remain imperishable in the annals of medicine. In some respects this statement cannot be altogether denied. At the same time, when we glance over the history of our profession in past and present times, and review the really great discoveries which have influenced its progress or established our knowledge, it is questionable whether that country is entitled to more than an equal share of honour. In the investigation of the neuroses it may be admitted that the French have taken a specially leading position, and this may be explained by the fact that this class of disorder is more prevalent, more widely distributed, and presents more severe forms, than with many other nations. It would, however, ill befit those regions of the world which are more fortunate in the comparative absence of such nervous maladies, to grudge any credit to their confrères for the advantage they possess in a larger experience, which they have so eminently utilised. They therefore welcome a work like the present, which indicates the desire, common to all truly scientific, to advance knowledge, and extend practical usefulness.

A. Hughes Bennett.

The Applied Anatomy of the Nervous System.—By Ambrose L. Ranney, A.M., M.D., Adjunct-Professor of Anatomy, New York, &c. pp. 500. 8vo. H. K. Lewis, London, 1881.

The purpose of this work is indicated by the full title "The Applied Anatomy of the Nervous System, being a study of this portion of the human body from a standpoint of its general interest and practical utility, designed for use as a text-book and a work of reference." Works of this nature have appeared only within a comparatively recent period, and the necessity for them becomes gradually more manifest as anatomy and physiology become every day more and more of independent sciences. To the last generation of medical men, anatomy meant almost entirely

surgical anatomy, and even yet there lingers something of that tradition. Such a work as the last edition of Quain's Anatomy is a magnificent protest against such a notion, doing full justice as it does to the anatomy of the nervous system and of the thoracic and abdominal viscera. But the old system, notwithstanding its narrowness, had its good side. What anatomy was retained had a practical bearing, and exerted, so far as it went, its due effect on the practice of medical men. It was all applied anatomy. Can we always say this at the present time? We fear that the rank and file of students, those namely who are to form the bulk of our practitioners, fail to see any practical bearing in the greater part of the anatomy and physiology of their first two years of medical study. As a consequence they do not digest and assimilate these subjects but simply swallow them, more or less completely as the case may be, with a view to successful disgorgement at their examinations. The process is fatal to anything like a systematically scientific diagnosis and treatment of disease. The evil has been recognized, and works more or less allied to the present have been published in the various departments of anatomy and physiology, their avowed purpose being to serve as links between the abstract sciences and their concrete embodiments in clinical medicine and surgery.

Detailed criticism of such a work as this, is of course impossible, containing as it does an immense number of facts, theories and explanations. Criticism is, however, much simplified by the fact that there is no claim and no attempt at originality or even at independent observation. The author has found an abundant material ready to his hand, and the question therefore resolved itself into one of clear and accurate statement with methodical and perspicuous arrangement. He has succeeded on the whole in producing a readable book, the diffuseness and iteration of which results no doubt from its having been originally delivered in the form of lectures before the medical students of New York University. Throughout the book the author makes very large use of Hilton's classical work on rest and pain. The principle of a common nerve-supply in cases of localized pain associated with distant disease, is one of immense importance in diagnosis and treatment; and its systematic and explicit recognition by Mr. Hilton constituted a distinct advance both in neurology and in practical medicine. At the same time it is well that the student who reads this work or Mr. Hilton's should remember that the nerve-fibres within a nerve have as little direct connection as a couple of telegraph wires that run on the same posts. We have got an important, a most important practical fact, but we are in reality no nearer the explanation of these strange cases. It must necessarily be a matter not of nerve-fibres but of nerve centres.

The first fifth of the book is devoted to the brain. It is somewhat wanting in cohesion and arrangement and is frequently by no means clear owing to a confusion of nomenclatures. For example the supra-marginal convolution of Turner is also called the supra-sylvian convolution and the parietal lobule, while the

term supra-marginal convolution is also applied to the portion usually termed superior parietal lobule. Functions and centres are discussed first, then topography, and finally functions and centres again. To the important subject of the vascular supply of the brain there is only the most meagre reference.

The two hundred pages devoted to the cranial nerves form unquestionably the best part of the work. The account of the innervation and movements of the eye-ball and specially of the iris, along with their clinical associations, is clear and full, although in a work like this we are somewhat surprised to find no reference to Charcot's cerebral decussation of the optic nerve-fibres. Again the anatomical and clinical discussion of the facial nerve is most detailed, but we find no notice of the peculiar character of the facial paralysis usually occurring in hemiplegia. Considering the importance of the principle and the light which it throws on many cases, we should have expected a full discussion of Dr. Broadbent's principle of the bilateral representation of muscles acting bilaterally, or as we ought rather to express it with Dr. Hughlings-Jackson, the bilateral representation of bilateral movements. At pp. 245 and 265 it seems to us that the efferent inhibitory action of the vagus is confused with the afferent depressor action.

The last part of the work is devoted to the spinal cord and spinal nerves, and is upon the whole both full and accurate. We should have liked to see a careful analysis of the various reflexes used in testing the condition of the cord at different levels. English readers will do well to note that Dr. Ranney uses the term postero-lateral column to mean the posterior part of the lateral column, not as is usual the lateral or external part of the posterior column, sometimes called the root zone of Charcot.

The work is freely illustrated, principally by the beautiful plates of Sappey and of Hirschfeld. A few diagrammatic figures are added, some of which are really useful, but some are useless, and others, such as Fig. 65, are both useless and incorrect.

As already indicated we approve most cordially of the purpose of this work, and doubtless in a second edition Dr. Ranney will by condensation and addition much improve its present form.

JAMES ANDERSON, M.D.

Lehrbuch der Neurologie.—By Dr. G. SCHWALBE, Professor of Anatomy at the University of Königsberg. pp. 740. 8vo. Erlangen, 1881. Eduard Besold.

THIS work, which forms the second part of the second volume of Hoffmann's 'Human Anatomy,' has been for some time in the hands of neurologists, and perhaps the best criticism passed upon it is the fact that we are already familiar from our recent English

text books with many of the original observations and wood-cuts that appear in it. The first forty pages are devoted to an introductory account of the nervous system generally, with a description of the minute anatomy of nervous tissue. Terse, clear and pointed, with all the advantage of a good typography, varied so as to express to the eye the importance of the different heads, this introduction is a fair specimen of the rest of the work. A short section on the methods of neurological research is specially valuable and interesting. These methods he divides into morphological, physiological, and pathological. Under the first head he discusses the purely anatomical method, showing the restrictions under which this method labours in the case of the nervous system, and the great assistance derived by it from comparative anatomy and from development. The physiological methods of section or extirpation and stimulation are next referred to, leading naturally to the experiments performed for us by nature in disease and the results of disease or injury, including the Wallerian degeneration and its interpretation.

Passing on to the spinal cord, we are at once struck by the fact that while the work is strictly anatomical, it still, as we should expect from the introduction, takes full cognisance of the results of physiological and pathological research. The result is, that this work, more than any other on the anatomy of the nervous system that we have seen, can be most cordially recommended as a suggestive guide to those engaged in the study of nervous diseases. A marked feature of the work is, that at the end of each section we have a copious bibliography well up to date, a point that will be, we doubt not, fully appreciated.

The account of the brain and its structure, with the deep origins of its cranial nerves, will be at once recognised as the most important part of the work. As is now customary, the developmental rather than the anatomical arrangement is followed. Any inconvenience arising from this is easily overcome by a short preliminary account of the anatomical subdivisions. This part is enriched even more than the rest by numerous original plates, showing vertical as well as horizontal sections of the brain. Considering the great practical importance and also the difficulties in interpretation of the physiological experiments of Fritsch, Hitzig, and Ferrier, Professor Schwalbe's chapters on the variations and the comparative anatomy of the cerebral convolutions, will be welcomed and read with interest. Specially important are the sentences with which he closes this section, "We are at the end of our comparison, without sure results for the comparison of the cerebral convolutions of primates with those of other forms. This, I believe, need cause no surprise when we remember that in the case of the two great groups of mammalia compared, namely, the Primates and the Carnivora and Ungulates, we have to do with divergent developmental forms. In the group of Primates we see development commence with almost smooth brains, which in their collective faculties nevertheless stand incomparably higher than the brains of carnivora and ungulates. In each of the groups then

there appears an independent convolution of the cerebral cortex on an already markedly different ground-form. It is therefore impossible accurately to compare the Primate brain with other mammalian convoluted brains, and I believe that for this reason the attempt to find the fissure of Rolando in one or the other fissure of the carnivorous or ungulate brain will have but little successful outcome." It is in the doctrine of evolution and dissolution, as applied psychologically by Herbert Spencer, and physiologically by Dr. Hughlings-Jackson, that we possess a clue to the mazes of mental and cerebral development. But in applying the doctrine we have to bear well in mind that we are dealing not with a ladder, step above step, but with a tree from whose trunk branch after branch strikes off.

The most valuable and most original part of the work is that devoted to the medulla oblongata and the great cerebral ganglia, with their relations on the one hand to the spinal cord, and on the other to the cerebral cortex. The numerous and beautifully executed wood-cuts introduced to show the structure and relations of these parts, constitute a most definite and valuable addition to neurology. Here again the statement of physiological and pathological results alongside of, but in no way confused with, anatomical facts, will be found specially valuable. In passing we may refer to the exhaustive bibliography of literature on the brain, to be found at p. 768.

While the last two sections of the work, devoted to the cranial and spinal nerves in their course and distribution, show fewer marks of original work than the preceding, they contain a full and accurate account of these nerves, written in a clear and easily followed style, and well illustrated by the plates of Hirschfeld and Leveillé.

We cannot but think that a mistake has been made in rendering this work incomplete through the omission from it of the vascular system of the brain and spinal cord. For this we are referred elsewhere, and perhaps it was unavoidable in view of the fact that the work before us forms only part of a large systematic treatise on Anatomy. Something is done to fill the gap by a few pages giving the blood supply of the various divisions of the nervous system, and also in the description of the meninges. The work would be very greatly improved by the addition of an index, which would add much to its utility as a work of reference.

JAMES ANDERSON, M.D.

The Field of Disease, a Book of Preventive Medicine.—By B. W. RICHARDSON, M.D., LL.D., F.R.S. 1 Vol. 8vo. London, 1883. Macmillan & Co.

UNDER this title Dr. Richardson has addressed a bulky volume of 1014 pages to "the intelligent reading public," in the belief that they "wish to know the leading facts about the diseases of the

human family." In accomplishing his task the author has been careful, as he tells us, to "avoid the new names and terms which have recently stolen their way into the literature of medicine, preferring the old historical terms as not only familiar, but far more correct than the new ones." It is a bold resolve, and whatever may be thought of its wisdom in general, is certainly unfortunate in the present instance, inasmuch as it invites attention to some of the chief blemishes of Dr. Richardson's book. The old pathologists, in spite of their metaphysical notions and fantastical nomenclature, were graphic and accurate describers of clinical symptoms. "The Field of Disease," on the contrary, is neither graphic nor accurate in this particular. To speak only of the department of nervous disease, which is neither better nor worse than the rest, it is surely but a lame description of apoplexy to say that "it means a blow or sudden shock by which a person is stricken down," adding no further account of its paralytic symptoms than the statement that it is "followed by some deficiency of power or partial paralysis, which may be permanent, but from which there is often a fair recovery." Again, it is somewhat vague to speak of sclerosis as a "condensation of the brain substance, extremely similar to what has been before us when treating of the disease of the lungs, known as fibroid disease." What is added regarding its symptoms, does not contribute to any more precise notion of its natural history or modes of incidence. "It may come on in early life, it may come on in later life, and it may be one of the degenerations of old age. When it is present in children they lapse into imbecility. When it attacks the aged it leads to failure of the vital powers and to the second childishness and mere oblivion."

But it is not always that the book is thus uncertain and featureless. Witness, for example, the account of anæmia of the brain as differentiated from congestion. The question involves so nice a point of diagnosis, that the passage may be worth quoting. "Congestion of the brain," it appears, is "due to excitement and over mental work, as also to alcohol, passion, worry, and excessive study. The opposite condition, known as anæmia, is produced by mental and physical exhaustion, by insufficient food, by long continued physical work and fatigue." Striking out, therefore, the factors common to both—for over-mental work and excessive study may be taken as the equivalent of mental exhaustion and fatigue—we have excitement, over-mental work and worry combined with alcohol to produce congestion, while physical exhaustion together with insufficient food produces anæmia. We are further told that while anæmia produces ringing sounds in the ears and somnolency, congestion, "in its milder and more common forms," (forms, that is to say, of which, by the hypothesis, morbid anatomy knows nothing), "gives rise to a sense of fulness in the head, dizziness, restlessness, and desire for change, with sleeplessness and irritability of mind," a condition which "often repeated may lead to diabetes or paralysis." It may sound very probable, but is not all this the pathology of the fireside, rather than of the post-mortem room?

Much more might be quoted to like effect, the description of tubercle, of meningitis, of cerebral softening, of functional nervous disease. Each successive page is a new surprise. As we read of chorea, that it is a disease "whose phenomena present themselves at all ages," (with no mention of children or of females as its peculiar subjects), and are informed that "recovery sometimes occurs;" or encounter the striking statement, that the pleura is "that membrane which can so easily be stripped from the surface of the lungs," it is difficult to withhold a suspicion that the author himself is hardly as familiar as he should be with some of the matters he undertakes to simplify. And yet we are assured, more than once or twice, that it is from Nature herself that Dr. Richardson is drawing his descriptions. He has not only discovered a new form of phthisis "peculiar to those who indulge in alcohol," and which (without describing it anatomically) he proposes to call "alcoholic," but chance once threw in his way a quite unique case, which he uses as his warrant for the rather bold statement, that "extreme congestion of the pia mater is a cause of delirium tremens." It is so characteristic of the author, and of the sketchy, anecdotal style of his book, as to be worth quoting. A man committed suicide by casting himself under a railway carriage. "Within four minutes after death," Dr. Richardson saw that man's pia mater "lying like a velvety pile over the brain, and within the convolutions, while still the fumes of whisky were most readily detectable from it." Nothing is said of the grounds of the diagnosis, the nature of the injury which thus laid bare the pia mater, or of the post-mortem examination which so speedily followed. Only one bold touch is added. "In such examples the membrane, after many excitements, becomes firmly attached in parts to the substance of the brain."

It may be truly urged, that to give a faithful description of the facts of medicine in popular phrase is a task of no small difficulty. But here one is reminded of the dramatic author, who when censured for the badness of his composition, and pleading in excuse that it was not easy to write a five act tragedy, was met with the reply that it was quite easy *not* to write a five act tragedy. And it is quite easy not to write a popular account of practical medicine, especially easy, it might be supposed, for Dr. Richardson, busy with other subjects where his authority is undisputed. The general reader is already surfeited with popular accounts, and weary of being studiously talked down to. For any information he may require on medical subjects he will do wisely to consult the very same sources of knowledge as attract professional students—the works of recognised teachers like Sir Thomas Watson, or even some such text-book as the admirable and lucid compendium of Dr. Frederick Roberts.

And, indeed, in the later part of his book, and when no longer concerned with the precise anatomical connection between lung and pleura or puzzling himself about the distinction between meningitis and acute hydrocephalus (p. 218), the composition is so clear and masterly that it is difficult to believe that the authorship

is the same. The part concerning "the cures and preventions of disease," contains much valuable information conveyed in simple and forcible language, which the best popular teachers may well envy. We have now no longer to criticise, but to listen; and read with amazement and concern of the anæmia, nervous depression, dyspepsia together with various derangements, circulatory and respiratory, which flow from the use of tea, coffee, tobacco and snuff. Not only, as it appears, is the cup that cheers, equally with the one that inebriates, laden with mischief for us, but the common emotions and passions produce distinct forms of disease. Intermittence of the heart, it seems, is *invariably* traceable to an emotional cause (p. 655), and even blushing (*erythema fugax*) is not so harmless as it looks.

In telling us that cheerfulness and contentment must be kept up, Dr. Richardson is but repeating what was said long ago by Mr. Squeers, but the wonder is to find the subject so closely studied that in "The Field of Disease" each particular passion has assigned to it its own peculiar danger, while articles of common consumption—and especially, be it noted, afternoon tea—are found to affect the human organism in ways but little suspected, producing, amongst other evils, nervous depression and low spirits.

Finally, and still with a master's hand, Dr. Richardson discusses the general principles of living in order that health may be preserved and old age postponed. We are not to grieve, not to hate, and not to be jealous. Yet, after all it is but a dreary picture, this passionless man, living in an evil and adulterating generation, mistrustful of the promptings of his special senses, for ever anxious and thoughtful about his own body and so encompassed by hidden dangers and subtle poisons that, with the very best intentions, Health can only be preserved by a diligent study of the voluminous literature which now goes by that name. Nay, it cannot be preserved anyhow in its hypothetical perfection; for the very concern and anxiety which this watchful regulation of self demands, is of itself a detriment and must in a measure defeat its own object. The smoker of tobacco and the afternoon tea-drinker may be sad spectacles enough, but sadder still, I think, is the contemplation of the man who refrains from emotion or sympathy on the score of his digestion, and with no better end in view than the prolongation for a little of his own lean and self-regarding existence, is continually worrying himself and his friends about the things of his body, the purity of his food and his drink, or the dictates of prudence in regard to a second cup of tea. The life is more than meat and the body than raiment.

OCTAVIUS STURGES.

Abstracts of British and Foreign Journals.

Pitres and Vaillard on Non-traumatic Peripheral Neuritis. (*Arch. de Neurologie*, April 1883, p. 191.)—The microscopical appearances of peripheral neuritis have hitherto been determined almost exclusively from cases of nerve-injury, but the authors of this most important contribution, which with the previous observations of Déjérine and Leloir opens out a wide field of new enquiry, point out that spontaneous non-traumatic peripheral neuritis is by no means an uncommon affection. It has origin in exposure to cold, in lead poisoning, variola, diphtheria, and typhus, in tuberculosis and alcoholism.[1] The extent of its domain is increasing daily, and it appears certain to occupy a position of considerable importance in the future. It has fallen to the lot of these observers to meet in ordinary hospital practice with a number of cases of peripheral neuritis independent of injury, and they have lost no time in placing their observations on record. It is a point of some moment that in the cases seen by them the ordinary or naked-eye appearances of the nerves presented nothing abnormal, and if to this fact be added the belief that inflammation of a nerve could not arise without some previous central change, it is no wonder that diseased nerves should have been frequently passed over in post-mortem examinations. The histological changes of spontaneous neuritis much resemble those seen in the peripheral portion of a divided nerve, although lacking the regular uniform march of the true Wallerian degeneration. The

[1] It has, however, been suggested by De Watteville that the peripheral changes of lead paralysis, and perhaps of multiple neuritis, were due to a dynamic disturbance of the spinal centres, interfering with their trophic influence ("The Pathogeny of Lead-Paralysis," 'Lancet,' 1880). Erb ('Brain,' 1883) appears to have independently arrived at the same conclusion, and develops this view in a paper on "Certain forms of Neurotic Atrophy" (*Neurol. Centralblatt*, 1883, No. 21). His chief conclusions are that, 1st, in presence of degenerative atrophy in nerves and muscles, the absence of spinal histological changes does not prove the absolutely peripheral nature of the affection, and the non-participation of the trophic centres; 2nd, that the fact of the motor roots being healthy does not prove the peripheral origin of the changes in the nerves.

rate of progress varies materially in different cases, being some times slow, at other times very rapid. From the observations already made, it seems to the authors probable that in some cases the neuritis may extend from periphery to centre, or attack by centripetal migration the spinal ganglia, and even the spinal cord itself. They recognise five different phases or types of degeneration, for an account of which it is needful to refer to the paper itself, and to the beautiful coloured drawings which accompany it. The types are thus described:—I. Fragmentation de la myéline en blocs. II. Fragmentation de la myéline en boules. III. Fragmentation de la myéline en fines granulations avec atrophie discontinue de la fibre nerveuse. Etat variqueux, moniliforme. Renflements fusiformes. IV. Atrophie des tubes avec granulations ambrées dans l'intérieur des gaînes. V. Atrophie complète des tubes nerveux; vacuité des gaînes: headings which very aptly describe the characters of the degeneration. The authors then give a full account of four cases, in three of which there was acute bed-sore—the "decubitus acutus" described by Charcot—after hemiplegia, and in which the peripheral neuritis was well marked. It is a striking fact that in the first of their cases, and to a less extent in the others, although the patient only survived the apoplectic attack six days, the nerves in the neighbourhood of the bed-sore were profoundly altered, even to almost complete atrophy of their fibres. Wallerian degeneration never runs so rapid a course as this. Their last case is one of zona in the sixth and eleventh right intercostal spaces. The periphery of the eleventh nerve was found diseased throughout its entirety, while of the sixth nerve the fibres of the root and ganglion as well as the periphery were extensively degenerate. These observations are continued in later numbers of the same journal (May, p. 290, Sept. p. 180), the authors recording the clinical history of several cases, together with their microscopical examinations. Discussing the ætiology of Zona, they are of opinion that too much stress has been laid upon the root and ganglion changes and that microscopical examination has not been sufficiently directed to the nerve periphery, alteration in which was the one common pathological change in their own case of diseased 6th and 11th nerves. In all probability they think that the degeneration of the ganglion and of the posterior root, found in the 6th nerve but not in the 11th, was secondary to the peripheral inflammation, and that this latter it was which caused the herpetic eruption. Microscopical examination thus supports the view, which has been

frequently expressed, that Zona may be due to a peripheral neuritis rather than to any central change. Cases VI. and VII. are most instructive examples of trophic and joint changes occurring in the course of tabes dorsalis—locomotor ataxy—and the authors call prominent attention to the fact that these changes were in each instance found to be associated with morbid states of the peripheral nerves supplying the affected parts. It had previously fallen to their lot, they relate, to make a careful examination of the spinal cord in cases of this kind, without being able to discover any pathological changes whatever in the anterior cornua, where by some it has been thought that the morbid condition may lie on which such trophic changes depend. They discuss at length the various reasons for attributing the arthropathies, and other trophic disturbances, of tabes dorsalis to central disease, and from a consideration of their own observations they conclude that it is at least highly probable that alterations in the peripheral nerves supply the real cause. At any rate it is a remarkable fact that in Case VII., where numerous lesions of nutrition arose in the course of tabes, to wit perforating ulcer, dystrophy of toe-nails, and arthropathic changes in a metacarpophalangeal joint, the peripheral nerves supplying the parts affected were the seat of extensive degeneration. That their observations are as yet too few to establish any precise dictum on this point the authors freely admit, and their conclusions are both cautious and provisional. All peripheral neurites do not of necessity give rise to trophic changes, but yet it is very obvious that trophic changes are in all probability provoked or caused by some primary alteration in the nerves. In Case VIII. there were developed numerous bed-sores, consecutive on myelitis from compression, which rapidly closed the scene in an old-standing case of angular curvature of the spine. The periphery of the nerves going to each one of the many sores was found atrophied and almost completely destroyed. But the point which above all they look upon as interesting in the examination both of this case and of others is the observation that the degeneration, extreme at the periphery, lessens gradually in degree as it is followed up the nerve-trunks, and that there is no evidence whatever of a continuous change extending in the reverse direction. And in those instances where the degeneration spread high up, the posterior roots were manifestly changed, although nothing abnormal whatever could be found in the anterior. The authors refrain from offering any explanation of this difference in the anterior and the posterior roots; they are content to record the fact which they have observed.

Thus, in their two somewhat analogous Cases, VIII. and IX. of myelitis dependent or spinal caries, with various trophic lesions in the last days of life, there were found well-marked changes in the posterior roots but none in the anterior roots of those nerves whose peripheries were diseased. How a lesion of the cord can exercise an influence on the periphery of nerves, and yet spare the trunks of these nerves, it is not easy to explain. One might assume that the degeneration spread continuously from centre to periphery, but the facts do not warrant this conclusion; and the authors are disposed to think that central lesion somehow predisposes to, but is not an immediate cause of, the peripheral neuritis which may arise in the course of disease of the central nervous system. The central lesion, on this hypothesis, so modifies the peripheral nutrition, that inflammatory or degenerative changes in the nerves are liable to arise from some accidental cause, such as undue pressure or other trifling form of injury. Be that however as it may, there is a radical difference between the centripetal march of a peripheral neuritis arising in the course of cerebral or spinal cord disease, and those centrifugal, or true Wallerian degenerations, set up by transverse section of nerve-trunks. That non-traumatic peripheral neuritis is a by no means uncommon affection the authors feel assured, since in a very short space of time, and with no great abundance of material, they have found no fewer than nine examples. They point out that the naked-eye appearances of the nerves are wholly insufficient to tell us whether a nerve is diseased or not, for these are usually normal, even when the microscope may reveal extensive pathological changes. And these changes essentially affect the ultimate nerve elements themselves, and only secondarily and at a much later date do they implicate the connective tissue. Following a variable course, unlike that described by Waller, the degeneration may amount to almost complete atrophy in the course of a few hours or days; and while no continuous connection can be traced between central nerve-lesion and peripheral disease, the neuritis beginning at the periphery seems to follow a truly centripetal march. Such are the main conclusions in ths iinportant contribution which deserves the careful study of all who are interested in the diseases, and the ætiology of the diseases, of the nervous system.

Déjérine on the Peripheral Nerve Lesions of Ataxia and on Peripheral Nervo-Tabes. (*Comptes Rendus*, 1883.)—This is a further and most important contribution, by an observer whose

researches in the same direction are already widely known, towards a fuller appreciation of the changes met with in the peripheral nerves in the course of tabes dorsalis. Struck by the fact that although defects in sensation had been in several cases very various, the cord lesion was found in each to be the same, the author was led to make more careful examination of the cutaneous nerves, and the paper before us is devoted to an extended record of these two cases with complete clinical history and post-mortem examination. Both were well-marked instances of the disease, with ataxy, abolition of tendon reflexes, crises, and lightning pains, and in addition thereto were even definite areas of anæsthesia and analgesia of the trunk and limbs. The usual central changes were found, and microscopical examination of the cutaneous nerves led further to the discovery of grave alterations therein in the regions of impaired sensation. Thus in the first case we read that the nerve trunks presented exactly those appearances which are ordinarily seen some months after section, very few indeed of the nerve fibres having the normal physiological qualities. In the second case, that of a woman aged 55 who had pronounced anæsthesia of the legs, the cutaneous nerves showed extensive degeneration indicative of an essential parenchymatous neuritis. The anterior roots were perfectly healthy, but the posterior roots between their ganglia and the cord were markedly altered. Below the ganglia, however, that is, between them and the point of coalescence of the anterior and posterior elements of the mixed nerve, no such changes were to be found. And the ganglia themselves were also healthy, an observation on which M. Déjérine lays stress as an indication that the peripheral changes of the nerves were not the result of any morbid condition of their trophic centres. The author enters into a full discussion upon the nature of these peripheral lesions, combating the view that they are in any way allied to the changes seen in the optic nerves, and coming rather to the conclusion that they are the result of a true peripheral neuritis, starting and developing like the medullary lesions quite independently, and showing no connection therewith, either as cause or effect. Peripheral lesions are daily becoming more known, and although experimentally speaking true Wallerian degeneration has an existence beyond all dispute, it is none the less true that the cutaneous nerves may show serious degeneration without any central change, the known examples thereof becoming rapidly more numerous as observation is more frequently directed to their examination. In the present state of our knowledge it is

impossible to speak with any degree of certainty as to the mode of origin of peripheral neuritis, or as to the precise influence it has in giving rise to some of the symptoms of tabes dorsalis, the retardation of, for instance, sensory impressions, the incoordination, &c., but there can be little doubt that it does play an important part in the disturbances of sensation which are so common, though so variable, in the disease. And confirming and strengthening these conclusions are the facts recorded in a later and most interesting communication from M. Déjérine on two cases of "peripheral nervo-tabes," in which there were many of the symptoms of locomotor ataxy ('La France Médicale,' 30th of October, 1883, No. 31), pains, incoordination, abolition of knee-jerk, anæsthesia, analgesia, and retardation of perceptions, where post-mortem no central lesions whatever could be found, the cutaneous nerves alone showing disease, having in fact undergone extreme degenerative changes, the result of peripheral neuritis. These cases are now only briefly recorded, for M. Déjérine intends to bring them forward in greater detail in a work on which he is at present engaged, and which will be looked forward to with much interest. He has found nothing like them in medical literature, and he places them on record with the object of showing how in some cases locomotor ataxy may exist independently of lesion in the spinal cord, and of suggesting for them the name, "Nervo-tabes périphérique," in contradistinction to that of "Tabes médullaire."

Johnson on Nerve-suture and Nerve-transplantation. (*Nordiskt Medicinskt Arkiv*, Band xiv. No. 27, p. 1.)—This is a contribution to the study of nerve-suture, both from clinical and experimental observation. The fifty-two cases collected by the author from various sources, and arranged in an admirable table, speak very forcibly of the benefit usually derived from the suture of divided nerves. M. Johnson has himself conducted a large number of experiments in the laboratory of the school of medicine at Stockholm, both on nerve-suture and nerve-transplantation, his observation being chiefly directed to a knowledge of the time when conductivity was re-established after the operation. This varied in different animals, being found on the 40th day in rabbits, on the 31st in dogs, and the 25th in fowls. It was not, however, until the 60th day that conductivity was restored after simple division of the sciatic in rabbits, and when no suture was applied. This was tested in each instance by a weak faradic current, or by some mechanical irritation, applied above the cicatrix. Microscopical

examination, moreover, showed a like difference in point of time, nerve fibres being in rabbits clearly traceable through the cicatrix on the 40th day after suture, on the 60th day only after simple section. The fibres in the latter case seemed rather better developed than in the former. Regeneration took place with greater speed in dogs and fowls. M. Johnson points out that nerve-suture is free from danger, neither trismus nor tetanus having followed it in any case; and he strongly recommends the practice, there being no question that restoration of function is thereby accelerated. It is less painful, and less likely to set up trophic disturbance, if the suture is indirect than if it is passed through the nerve-trunk itself. In three cases, where pieces of the sciatic had been resected in fowls, he endeavoured to transplant portions of nerve, taken in two instances from the sciatics of other fowls, and in one instance from the sciatic of a rabbit. The animals were killed on the 28th, 24th and 23rd days respectively, but although the grafts were in each case firmly united, conductivity was not the least established, and, as might indeed have been expected, microscopy revealed almost complete degeneration of nerve-structure in the engrafted fragments, in marked contrast to the condition of the nerve itself. In addition to the large number of collected cases, the paper is enriched with an account of other experimental observations, and with a full bibliography on the whole subject of nerve-injury and nerve-suture. HERBERT W. PAGE.

Two Cases of so-called Spastic Spinal Paralysis. In a reprint from *Il Movimento medico-chirurgico*, Professor Bianchi details two interesting cases, and gives an account of the changes found in the nervous system after death. The first case was that of Giuseppe Ippolito, aged 67, who in his occupation as a baker had been exposed to sudden changes of temperature. Had been a soldier, and been punished altogether to the amount of 830 blows. At 25 years of age had syphilis. In 1852 felt a weakness of the lower limbs with rigidity, worse on rising in the morning, or on getting up from his chair. Had to use first a stick and then a crutch to get about. No other ailment, except slight formication. By-and-by tremblings in the limbs on walking so that he required two crutches; and loss of power over the joints, with extreme rigidity. When he came under the notice of Professor Bianchi, he suffered from no loss of sensation nor any muscular atrophy, except a certain loss of rotundity about the limbs. The man lay in bed unable to move, the thighs flexed on the abdomen, the legs on the

thighs, the feet in slight dorsal flexion and abducted, the knees so tightly compressed against one another that he had to put a pillow between to prevent the suffering. The cutaneous reflexes natural, or rather increased. Electric examination showed no changes of importance. Before death changes occurred in the sensibility; and there was paralysis of bladder and rectum.

On necropsy, the spinal marrow was softened and reduced almost to a pulp from the lumbar region as high as the third dorsal nerves. On section the bundles of grey matter normal, except that the columns of Goll shewed a noticeable change of colour observed after the use of both hæmatoxylin and picro-carmine staining. On account of the great length of time during which the disease persisted (31 years) no certain conclusions are drawn by Dr. Bianchi from this case.

Case 2 is more important. It is that of a boy, G. Finorelli, aged 12, a deaf mute of deficient intellect. At 9 years of age began to be unsteady in his gait, his steps being constrained and the body inclined to right or left, in order to enable him to lift his feet, the points of which he dragged along the pavement. When he rested on the ground, the lower limbs trembled, and when he got into bed, they became rigid. The disease made rapid progress, and soon the boy could not walk at all, though he used his hands perfectly. No rigidity of the superior extremities, but lower limbs rigidly contracted. Soon, however, difficulty arose in using the left upper limb which was followed by complete immobility. Two or three months later, rigidity of right upper extremity. At this time Dr. Bianchi had the opportunity of watching the patient. Spasms of the trunk and contractions of the muscles of the neck followed. The lad seemed to have pain, for he cried out when strong contractions of the lower limbs occurred. The lad now lay always supine, the lower limbs immovable and rigid as bars, the muscles hard and raised. The rigidity which was greater on the left side, increased, the leg being forcibly extended on the thigh, the thigh on the pelvis, and the foot in the equino-varus position crossed over that of the other side. The first phalanges of the toes extended on the metatarsus, the second and third strongly flexed. On attempting to make movements, they could not be executed, and rigidity was increased, the stiffest muscles being the quadriceps extensor, the gastrocnemius, the posterior tibial, and the flexors of the toes. No tendon-reflex when the limbs were rigid, but when a little relaxed the patellar-reflex was increased, and sometimes knee clonus came on. The cutaneous reflexes became diminished. The cremasteric

reflex nearly disappeared. At the same time tickling of the sole increased the contraction, the lad cried at the same time flexion occurred. When the coverlet was raised, the contraction increased, till it became painful, and drew cries from the lad. It was very difficult to judge how much the tactile sensibility was affected, the boy being dumb and unable to move. The left upper arm was forcibly laid along the side of the thorax, the forearm pronated and firmly flexed on the arm, the fist extended, the fingers flexed, the thumb folded in the fist, and the proximal phalanges extended on the metacarpals. The muscles well nourished, the biceps hard. The right upper limb could be somewhat moved, but efforts to do so required much more force than natural.

On electric examination, the muscles could be made to contract in the lower limbs by the induced current, but the contractions so induced did not overcome the antagonistic contractions of the muscles in spasm. The induced current also caused the muscles to contract in the upper limbs, but on the left side every application of the current on the forearm increased the rigidity of all the muscles contracted in mass, and from the position of the limb it was not found possible to act on any of these.

By the galvanic current results were got from 12 to 26 elements, the fewest elements being required on the right side. Eight to ten days before death, the contraction of the muscles of the neck increased, piercing shrieks were emitted, passed along as it were from tract to tract; the temperature rose, the face became glassy and expressionless, some attempts at vomiting ensued, slight strabismus with somnolence, coma and death.

On necropsy, two facts bearing on the deficiency of intelligence were observed. The first was the incomplete development of the temporo-sphenoidal lobe, especially of the first temporal convolution. The second was the prolongation of the parieto-occipital sulcus on the outer surface of the hemispheres, through the defect of development of the first fold of the passage of Gratiolet, very much as is observed in the brain of certain monkeys. The occipital lobes were little developed, but there was an exaggeration in the development of the parietal lobes, so that the brain appeared short and high, and the fissure of Rolando less oblique.

The spinal cord cried somewhat on section by the knife. When ections were hardened for three weeks in solution of bichromate of potash and coloured with picro-carmine, two sets of alternations were observed. One of these was precisely and systematically limited to the pyramidal bundle of fibres of the two sides: the other,

without relation or apparent continuity with the former, was limited to the central canal and its surroundings. In drawings from a series of sections of the cord through the medulla oblongata at the middle of the olivary body, at the bottom of the olivary body, between the 1st and 2nd pairs of cervical nerves, at the level of the 6th cervical nerves, in the dorsal and the lumbar regions, the degeneration of the fibres making up the pyramidal bundle was consecutively traced, as also the relations of the same at different levels. From these it is seen, that the degenerated spot which was pretty large in the lumbar region was reduced considerably in the cervical, by the crossing over of some of the pyramidal fibres to the outer side of the fissure. The direct cerebellar bundle of fibres was also larger there. In the medulla oblongata no lesion of any moment was found, except in the pyramidal fibres. Figure 6 is very instructive, showing, as it does, in the section of the right side of the medulla oblongata at the middle of the olivary body, that the only abnormal portion is the anterior pyramid.

A section of the brain shewed a sclerosed nucleus of a grey yellowish colour, much harder than the surrounding substance, of the size of a bean, in the very centre of the centrum ovale, of which only a very small zone remained normal and divided the sclerosed nucleus from the cortical grey substance. This nucleus was prolonged backwards, and was found to extend to the fibres of the 2nd and 3rd frontal convolutions. Still further back it occupied the expansion of the *corona radiata* of Reil, corresponding with the ascending frontal and parietal convolutions, and was prolonged into the internal capsule which was degenerated in the posterior half of the anterior segment, the *genu* and a large part of the anterior portion of the posterior segment. A similar lesion was found on the other side, the degeneration being confined to the motor zone, and the nuclei of the base and the grey substance being entirely spared. In short the motor portions were sclerosed and the sensory portions were normal. Histologically, the degenerative changes consisted of an enormous increase of the connective tissue, developed entirely at the expense of the nerve-tubes; of an increase of the nuclei and of the lymphatic corpuscles; of a diminution of the nerve-tubes, many of which were, however, well preserved, some having the axis-cylinder most minute, and others the nerve-sheath sclerosed with a great diminution of the medullary sheath and the axis-cylinder much increased, these last however being rare.

In the internal capsule, however, a different appearance was

observed. Where the degeneration was most advanced, there was observed the same fibrous connective tissue found in the pyramids, with scarcely any nervous fibres for long tracts.

In the intermediate zone there were seen in the midst of a granular quasi-amorphous tissue an immense number of lympathic corpuscles and nuclei, with a development of capillaries, and no nerve-fibres. After further description of the changes found, Professor Bianchi makes some remarks on Schulz's question: Is there a primary sclerosis of the lateral columns of the spinal cord? and refers to a case published by Zucha to show that the symptoms may all be present without a lesion of the pyramidal bundles, the disease being confined to the brain. For this reason Dr. Bianchi suggests that the disease should be called *spastic paralysis*, and not *spastic spinal paralysis*. On this we would remark, that if it were called *sclerosis* of the *pyramidal tracts*, or sclerosis of whatever portion of the brain may be affected, or both, we should have a really scientific name of the disease. *Paralysis* is no more the name of the disease than *dropsy* would be of mitral incompetence, though each is a symptom in its own place. If there were any softening, that ought to be named also.

La Psichiatria, la Neuropatologia e le scienze affine is the title of a new quarterly journal devoted to inquiries in the domain of the nervous system. It is under the directorship of Professor Buonomo, medical director of the asylum at Naples, and is edited by Dr. Bianchi, assisted by Drs. Andriani, Armanni, Cantarano, Fede, and Nicolucci. This journal will take the place for the Southern Italian Provinces filled by the *Archivo delle Malattie nervose* of Milan, and the *Rivista sperimentale di Freniatria* and other journals published in North Italy.

Professor Buonomo leads off in the first number with an article on the value and importance of a study of nervous diseases, pointing out that the general acceptance of the theory of evolution has revolutionised this (as indeed it may be said to have done all other) department of inquiry, so that human psychology can no longer be the result of the self-introspection of the philosopher, but must be founded on comparative anatomy and comparative physiology. Professor Buonomo goes on to say that insanity is the special study of the medical psychologist, and to remark on the vast importance of its sphere in determining questions connected with mental capacity and responsibility, more especially in connection with penal legislation and civil legal relations generally.

Professor BIANCHI has an interesting and important article on *Functional Compensations of the Cerebral Cortex.* The various theories of accounting for these are considered shortly, and Dr. Bianchi goes on to formulate certain conclusions, detailing the experiments on which he rests them. The subject is continued in the second number of *La Psichiatria*, some 40 pages of which are devoted to it. The monograph is well worth reading, as was to have been expected from so enthusiastic an enquirer, but our space allows us only to state his main conclusions. The first proposition is the following: Recovery from the paralysis consequent on an extensive destruction of the motor zone in dogs is only apparent and partial, only locomotion and acts correlative therewith being restored; every other movement in different conditions remains permanently abolished (for months) and power is permanently diminished.

2nd. The parts surrounding the excitable points of the motor zone of limbs (which portions have remained perfect after a limited destruction) may to a certain extent compensate the functional power of the part destroyed, though without acquiring electric excitability when acting on the parts whose centre has been removed.

3rd. Locomotion is not determined by special centres in the brain of dogs; in these animals the cortical motor zone may be destroyed without any lasting disturbance in locomotion.

4th. In dogs, when the motor zone of one side has been widely destroyed, the opposite hemisphere, and especially its motor zone, exerts a controlling power on the limbs of both sides.

5th. If, during the period of development, the cerebellum has been in great part destroyed, the cortical motor zone may in dogs perform its functions.

Under the consideration of this proposition certain questions arise regarding the sense-organs, and Dr. Bianchi states the following conclusions:

(*a*) The fibres of the retina of each eye go in large part to the cortical centre of the opposite hemisphere, but to a smaller extent to that of the hemisphere of the same side.

(*b*) The cortical visual centre of dogs is very extensive, comprising all the second external convolution from its anterior extremity as far as the accipital lobe close to the latter, and part of the first and third external convolution.

(*c*) Compensation is made in the portion remaining entire when the cortical centre of the same side has been partially destroyed. It is very doubtful whether the centre of the opposite side contributes thereto.

In order to establish these conclusions some sixteen experiments are detailed, and under the head of general considerations, the two following propositions are laid down.

(A) A cortical paralysis is the graver, the more extensive is the lesion of the anterior lobes of the cerebrum, within certain limits not yet determined.

(B) The central elements of innervation for a given organ of motion are diffused over all the motor zone, and are merely found more condensed in certain points, which constitute therefore the so-called excitable zones, which up to the present time have been erroneously considered as the only cortical centres corresponding with special groups of muscles or with a given limb.

Thirteen corollaries which Professor Bianchi believes himself justified in drawing from these premisses, bring to an end this valuable paper.

Professor NICOLUCCI contributes an article of some merit on *the brain of man, considered under an anthropological aspect.* After shewing that according to the recorded nomenclature, there are 44 convolutions in the brain, and that 16 of these are in the frontal lobes, he says this plainly shows the great predominance, exerted by the frontal lobes over the other lobes of the human brain, and consequently the highly important part they play in the exercise of the encephalic functions. But Professor Nicolucci's inquiries have led him to results far less certain than those commonly believed, and he says: "I do not hesitate to declare that," the conclusions generally accepted regarding the localisation of the cerebral functions "are yet very doubtful, and even to some extent erroneous. It is also to be remembered that the experimental investigations of this complex question have been only commenced, and it is reserved for future observations to assist in forming conclusions less uncertain and more trustworthy." The writer then goes on to shew that observations are very deficient, up to the present time, regarding the weight and quality and convolution-markings of several human races, as for instance, Negroes, Arabians, Algerine Berbers, Chinese, &c. So far, however, as the facts are known, very great differences are found in the weight, and even in the disposition, of the substance of different human races; and these, in Professor Nicolucci's opinion, have not received the attention they deserve.

The remainder of the first two numbers of *La Psichiatria* is made up of numerous reviews. A. RABAGLIATI.

Archives of Psychological Medicine and Neurology. Kharkof, 1883. Parts 1 and 2.—It is significant of the lively interest excited by recent advances in the domain of neurology that a new quarterly journal, devoted to this subject, should make its appearance in a provincial town of Southern Russia. Great credit is due to the energy and initiative of its editor, Dr. P. Kovalevsky, who has succeeded in filling his two first numbers with much interesting material. As the journal is printed in the Russian language, its circulation will, of course, be limited to the Russian Empire. Any matter of striking originality or importance that it may contain is sure, however, to find its way to us through Germany. A brief summary of a few of the principal contributions to the two parts already issued, will serve to give the readers of BRAIN some idea of the scope of the undertaking.

The editor describes two cases of old-standing destruction of the eyeballs associated with wasting of the occipital lobes. In one, the right eye was alone affected, having been destroyed by an injury some twenty years before the patient's death. There was conspicuous asymmetry of the back part of the hemispheres, owing to atrophy of the convolutions of the right occipital lobe. In the second case, both eyes had been withered for more than twenty years, and the wasting of the brain was bilateral.—Dr. Orshansky furnishes the results of an experimental inquiry into the effects of anæmia in modifying the excitability of the cerebral cortex.—A most interesting account is given of the public lunatic asylum of Bessarabia by the medical officer in charge of it. The state of things described is quite equal to that which formerly existed in some of our work-houses. The one hopeful feature is that it should be possible, in a country like Russia, to publish so frank and outspoken a report. Improvement cannot be far off when once the light of day is admitted into such dark places.—A clinical lecture by Dr. Pasternatzki of Warsaw deals with a remarkable example of major hysteria in an adult male, a cornet of cavalry, twenty four years of age. This patient presented the symptoms of complete hemianæsthesia, induced hypnotism and catalepsy, violent hystero-epileptic paroxysms arrested at will by pressure on a hysterogenic area, and all the train of extraordinary phenomena with which the writings of Charcot have made us familiar. He seems to have been cured by the application of cold water douches to the spinal column.—A considerable part of each number is taken up with abstracts of papers from foreign journals and reports of cases of medico-legal interest occurring in the Russian courts.

In conclusion, it is only fair to add that the importance and usefulness of this new periodical are not to be measured solely by what it may add to our scientific knowledge. Those who are in some measure familiar with the conditions under which it is produced, and the public to which it appeals, will recognise and welcome it as a new window opened towards the West. Every such window comes to admit more air and more light than those who throw it open can foresee. E. BUCHANAN BAXTER, M.D.

Archives de Neurologie.—In the November (1882) number of the 'Archives' Dr. GELLÉ commenced a series of articles which deal with the clinical history of Ménière's disease, and the diagnostic features of different forms of labyrinthine vertigo. He briefly describes the usual form of attack, and alludes to the clinical studies of the disease instituted by Vowry, Bonnenfant, Lhuissier, Léo, Féré, and Demars.

Ménière, Saissy, Moos, Politzer and Voltolini have described affections of the labyrinth, and especially lesions of the semicircular canals in cases of pronounced vertigo with deafness, chiefly in cases of cerebro-spinal meningitis. In Gellé's cases, apart from the auditory troubles and disordered equilibration, the patients enjoyed perfect health. In Ménière's disease the troubles of equilibration, the buzzing sounds in the ear, and the deafness cannot all be referred to lesions of the semicircular canals; if the patient becomes quite deaf, it probably indicates something beyond disease of these canals, and this becomes a certainty if the deafness remain after the disappearance of the vertigo. Deafness is a most frequent accompaniment and termination of Ménière's vertigo, and therefore in seeking for the pathogenesis of the chief features of this disease, the deafness and buzzing, we must look for lesions outside the labyrinth.

The physiological experiments of Paul Bert and others, taken with the fact that simple injection of the ear will cause severe vertigo in man, suffice to indicate that any sudden labyrinthine commotion, such as would be induced by an inward displacement of the base of the stapes, or the membrane covering the fenestra rotunda, will initiate the vertiginous state; and thus "experimental vertigo" may be induced by the pressure of a column of air upon the membrana tympani transmitted along the chain of ossicula towards the fenestra ovalis in those who are predisposed to vertiginous affections. It may be regarded as established that the troubles of equilibration in such cases depend directly upon the amplitude of movement of the stapes, or the immobility of the

membranous partition of the fenestra rotunda; either condition inducing increased pressure in the semicircular canals. Clinical data fully substantiate this view.

Upon examination of a case of labyrinthine vertigo, if disease of the middle ear be not detected it is usual to infer the existence of a lesion of the labyrinth; yet, to prove that deafness exists apart from lesions of the tympanic cavity is universally acknowledged a difficult diagnostic point, the difficulty consisting in exclusion of affections of the fenestra rotunda and ovalis. Dr. Gellé then discusses the methods of exploration of the labyrinth, and the employment of the diapason,* placed on frontal eminence. He regards the usual assumption that the vibrations are propagated *direct* through the cranial bones to the contents of labyrinth as an error; for he has long shown that sounds thus produced are greatly modified in intensity by altering the state of tension of the transmitting chain of ossicles. Savart and Wollaston have proved the rule to hold good for aerial vibrations. Experiment by centripetal pressure upon the tympanic membrane leaves no doubt upon this point, and decidedly contradicts the views of Bonnenfant, Triquet, Politzer, &c., that diminished auditory perception of cranial vibrations is a good differential sign between enfeebled hearing, and an obstacle to the reception of sonorous waves. It would be more exact to say that this loss of cranial transmission indicates an obstacle to the access of sonorous waves on a level with the fenestra ovalis and rotunda.

He then insists on the value of his test by centripetal pressure, a "diapason" being placed on the frontal eminence of the side to be examined whilst the orifice of the meatus is compressed. In healthy subjects the pressure gradually increased modifies the quality, and eventually extinguishes the sound so transmitted, and a gamut of sensations is thus formed, corresponding to variations in the centripetal pressure, really indicative of the degree of mobility and elasticity of the transmitting chain of ossicles, and especially of the state of the stapes and the fenestræ.

In disease no such modification of sound may be obtained on pressure, or any sound transmitted may be absolutely extinguished upon the *gentlest pressure*, and vertigo and buzzing sounds produced. The conclusion is evident; the transmitting ossicles are immobile and the sound is not modified, or the movement is so slight that the gentlest centripetal pressure renders the stapes immobile and the vibrations are wholly extinguished. The vertigo may be

* By a "diapason" Dr Gellé would indicate a form of tuning-fork.

caused by excessive movement of the stapes, or by immobility of the membrane of the fenestra rotunda.

The relationship of softening of the membrana tympani to Ménière's vertigo is then discussed; if in such cases the meatus be obstructed, the inward displacement of the tympanic membrane increases gradually, the stapes becomes fixed in a new position, and complete deafness, or at least no modification of sonorous waves, results upon centripetal pressure; *if the meatus be free*, the vertiginous and other morbid symptoms coincide with efforts at swallowing, sneezing and yawning. A case is then recorded where vertigo was induced by very gentle centripetal pressure, and where there was complete deafness both for aerial and cranial vibrations; and yet the membrana tympani and whole chain of ossicles except the stapes had disappeared as the result of suppurative median otitis. In this case the stapes was clearly seen *in situ*, whilst the fenestra rotunda was covered by a cicatricial firm structure. In all cases the labyrinth bridled by the fenestræ is at the mercy of any slight circulatory disturbance. Amongst the many causes of Ménière's vertigo may be cited spasmodic reflex of the tensor tympani under emotional influences due to sudden violent sounds. A typical case of the kind is quoted, in which the vertigo is only occasioned by a noise; such excessive sensibility to noises being explained as due to a fixture of the stapes, and its inward displacement resulting in undue compression of the labyrinthine contents upon the slightest contraction of the accommodative apparatus. Cases are referred to where efforts of attention were always attended by lessened acuity of hearing; here accommodative movements extinguished the transmitted waves.

Heat is another efficient cause of vertigo, some patients suffer only during the heat of summer. Fixture of stapes (as in sclerematous otitis), if accompanied by the least auricular catarrh, may induce compression of the labyrinthine nerves and vertigo.

If on centripetal pressure the transmitted sounds are neither modified, extinguished, nor accompanied by vertigo, we should infer that the stapes is immobile—a condition which may result from inward displacement of the whole transmitting apparatus from old lesions, such as external otitis; yet, the arrest of contripetal pressure may occur at the other end of the chain of ossicles, as in sclerematous states of the membrana tympani, or a bridle may connect one of the ossicles with roof of tympanic cavity; here the sonorous waves are transmitted to the labyrinth, but are no longer modified by pressure, and yet the *stapes is free*.

The second test proposed by Dr. Gellé is what he terms "transauricular auscultation, during centripetal pressure the vibrating diapason being placed on frontal eminence." The ear is examined by the otoscope, and the sonorous waves transmitted across tympanum can be heard by the observer and modified by centripetal pressure. Thus can we distinctly differentiate the distal from the proximal fixture of the chain of ossicles, and so correctly localise the lesion; whilst we check the patient's observations by those of the physician. An analysis of numerous cases of Ménière's disease follows, and as regards the vertiginous state the author concludes that it is an accident and not a necessary accompaniment of the auricular affections, depending on the idiosyncrasy, nervous temperament, extreme reflex excitability, rheumatic or gouty diathesis of the patient. It is especially liable to occur in pharyngeal congestions from obvious reasons. In the large majority of cases of Ménière's vertigo Dr. Gellé finds a grave lesion on a level with the base of stapes and the fenestræ. The author concludes a most elaborate and instructive series of articles by a review of the more important methods of treatment.

In the *Archives of Neurology* for March, Dr. PARINAUD calls attention to ocular paralysis of central origin, and particularly the paralysed condition of the associated ocular movements. Movements of the eye, like other voluntary movements, call numerous muscles into simultaneous action, requiring for this purpose a complete central system of co-ordination which pathological and anatomical facts reveal. Paralytic conjugate deviation of the eyes was described by Foville in 1858, the site of lesion being regarded by him as probably in the pons. Several cases were subsequently reported where this view was confirmed by the discovery of a lesion on a plane with the eminentia teres, implicating the nucleus of the abducens. Then came the researches of Duval, Laborde and Granx, which proved the existence in the cat of a connecting fasciculus betwixt the nuclei of the sixth nerve and that of the third nerve of the opposite side. Lastly, M. Landouzy has further evolved the question by his investigations of the relationships betwixt conjugate deviation and hemispheric lesion. The lateral deviation of the eyes whereby muscles supplied by different nerves are associated in action is probably explained by a similar mechanism to that occurring in associated movements of other parts. Duval has lately announced the existence of a nervous band which unites the nucleus of the

sixth and fourth nerves of the same side, and thus the nucleus of the sixth pair gives fasciculi to three motor nerves of the eye. In studying the innervation of associated ocular muscles it is therefore necessary to take into consideration this relationship of individual nerves to multiple centres of origin. The researches of Hensen and Welker indicate that the great nucleus of the third pair extending from the third to the fourth ventricle along the aqueduct, is really constituted of several distinct nuclei having relationships with the different muscles innervated by this nerve; whilst Vulpian showed that the reflex associated pupillary movements (consensual?) were dependent upon commissural fibres, since they ceased when a very superficial incision was carried along the median line of the floor of the fourth ventricle.

Beyond these connections there are the reflex paths uniting these nuclei with the optic nerves in the nates, according to the researches of Flourens, Ferrier, &c.; the influence of the cortex upon ocular movements has been established by numerous researches and observations; whilst the rôle played by the cerebellum, especially its inferior vermiform process, has been indicated by Duval and Laborde. Such complex relationships demand great caution in the interpretation of facts presented. Our author then proceeds to illustrate paralysis of parallel horizontal and vertical movements; and those of convergence and divergence. His first case is that of a young man, aged 27, who, after many months of severe headache increased by movement, was suddenly seized with sight troubles: in looking to the left, sight is blurred, but he never sees double. A difficulty is experienced in turning the eyes to the left, and there is a tendency to supplement the ocular movement by a deviation of the head instinctively to the left. Paralysis affects external rectus of left eye, and internal rectus of right eye, the former more marked than the latter, and both apparent for monocular and binocular vision. The internal rectus paralysed for parallel movements executes well movements of convergence. The conjugate paralysis is incomplete, there being no apparent deviation to the right when the eyes are in repose, but this becomes apparent after a fatiguing examination. By coloured glass we produce diplopia with all the characters of paralysis of the sixth nerve. Pupils equal, react normally. Visual acuity good; no lesion at fundus. No anomaly of sensibility or motor power in the limbs. Patient is emotional, trembling, and hesitating in speech. Deep reflexes preserved without exaggeration. A few days later vomiting supervenes, with vertigo and violent headache, especially

of right side; gait becomes embarrassed, with a tendency to fall to right side. Severe singultus lasting 48 hours, and then laryngeal spasms of some minutes' duration occurring five or six times through night, and threatening a fatal termination. Later on there is loss of power of right side of body. The dynamometer registers, right = 28 kilog., left = 48 kilog. Two months later there is appreciable deviation of eyes to the right, yet convergence is well executed and other movements remain normal. There is right hemiplegia and slight facial paralysis of the *same side*; no alteration of cutaneous sensibility. The instinctive deviation of the head to the left is instructive, and might at first view appear to be an exception to the law of Prevost as to the direction of the head in conjugate deviation of the eyes from lesions in the pons. The unusual implication of the facial on the side of the paralysed limbs, together with the laryngeal spasms, add interest to the above case.

His second observation is that of a lad aged 11, microcephalic, and suffering from chorea and congenital amaurosis, which had been relieved as he grew older. Visual field irregularly contracted; chromatic vision normal; atrophic decoloration of both papillæ. Patient is incapable of turning his eyes to the right, and looking to the left caused nystagmus; all other ocular movements are normal for monocular as well as binocular vision. Nystagmus is only present upon deviation of eyes to left side, and wholly disappears upon median fixation for distant vision and for convergence. Closure of one eye causes marked nystagmus in the other, rendering fixation in any direction impossible.

In the third case there is inability to turn the eyes to the right, the right external rectus and left internal rectus (more especially the former) being paralysed. Convergence is easy but exaggerated for the right eye, on account of the nearly complete paralysis of its abductor muscle. Deviation to left is still more readily accomplished, but is not of normal range; it produces nystagmus, especially of left eye. Elevation and depression of eyeballs normal and unaccompanied by nystagmus; never suffered from diplopia: no deviation of eyes is apparent in repose; no asymmetry of face. Has a tendency to rotate the head to right when he looks in that direction. Pupils equal, and contract normally. No lesion at fundus oculi. Amongst the other symptoms were deviation of the tongue to left and atrophy of its right half.

Dr. Parinaud proceeds to remark, that although paralyses of associated ocular movements are common in disseminated sclerosis, they are usually far less complete than in the cases just described.

The absence of diplopia in these cases often renders the state of ocular paralysis long undetected. The nystagmus, although not peculiar to *Sclérose en plaques*, is almost always associated with the conjugate paralysis dependent upon that pathological condition. Parinaud regards the nystagmus, like that occurring in early infantile amblyopia, as due to a lesion of nuclei in the grey matter superintending associated binocular movements. A difficulty in diagnosis may occur betwixt paralysis of the abducens, and paralysis of the conjugate movement initiated by the sixth nerve. In peripheral paralysis, however, of the outer rectus, the internal rectus of the opposite eye is the seat of exaggerated contraction; so that when the left external rectus is paralysed, if the patient looks to the left side, the healthy right eye deviates suddenly inwards, and fixation is accomplished by the paralysed eye. This differential sign renders the diagnosis simple. Nystagmus is of little import if it takes place in the direction of the paralysed muscles; but when in a direction opposed to these, it becomes a more significant symptom. Lastly, the absence of an alternate paralysis of face and limbs should call our attention to a possible conjugate ocular paralysis, not otherwise revealed by diplopia or persistent deviation.

His fifth case illustrates paralysis of parallel movements in the vertical plane: both elevation and depression of the eyeballs are abolished; lateral movements preserved, but convergence is impossible; the movements of eyelids are intact; right pupil largest; both moderately contracted and reflex contraction to light is abolished. The visual field is normal as regards form, light and colour. Cutaneous sensibility normal; no headache, vomiting or lapse of consciousness had occurred; a tendency to fall to the left side was observed, but is not due to vertigo; slowing of the pulse; urine slightly albuminous; no sugar; loud systolic bruit at base; no atheroma of radials. Recurrent polyuria with slight albuminuria should be considered in relation with the experiments of Claude Bernard, and probably indicate a lesion of the floor of the 4th ventricle, or a reflected irritation from some neighbouring site.

The tendency to fall to the left, with no appreciable paralysis of limbs, also refers us to the Rachidian bulb. As to the associated paralysis of the eye—if it affected but one eye, it might be explained by a lesion partially involving the nucleus of the third nerve, affecting *all the muscles of the globe* innervated by this nerve, but leaving intact the iris and levator palpebræ. Accord-

ing to the researches of Hensen and Welker, this lesion would implicate the posterior part of the nucleus near the upper angle of the fourth ventricle. The absence of paralysis of the internal rectus for lateral movements is readily explained by the fillet which the nucleus of the sixth nerve sends to this muscle.

The sixth observation deals with paralysis of the convergent movements of the eyeballs. An interesting case is quoted of asthenopia of the internal recti: convergence eventually wholly abolished; fixation producing strabismus; alternately covering each eye, the one uncovered executes sharply a movement of fixation; diplopia, once present, has gradually disappeared and can with difficulty be recalled by coloured glasses: *almost complete paralysis of accommodation without mydriasis;* pupils equal, moderately contracted; reaction to light very feeble and no reaction with attempts at convergence. Amblyopia characterised by irregular recession of visual field in both eyes, more pronounced in right eye on nasal side. With + 4 Diopt.: p. p. = 30 Cm.; no dyschromatopsia, no notable ophthalmoscopic lesions. Two months later, double optic neuritis; epileptiform seizures, leaving persistent trembling of arms, lip, and lower jaw. In this case a new growth was found involving the right crura cerebri and cerebelli, implicating the right optic tract, corpora geniculata and right nates and testes; right trochlearis is flattened and softened at its origin; and the right motor oculi is affected in like manner and split into four or five bundles by the pressure of the growth. The lesions were too extensive for an instructive localisation. Donders has shown that in the normal state convergence and accommodative effort is a synergic act. In the above case we see both actions abolished; but we must not conclude that accommodation would be *necessarily abolished* where convergence is impossible: we see the contrary in cases of strabismus, and when it occurs as a result of paralysis of the internal recti; accommodation may still exist, especially if the affection be gradual in its advance or of long standing. Dr. Parinaud concludes a very instructive article by a few further remarks on anomalies of movements of divergence.

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END OF VOL. VI.

LONDON: PRINTED BY WILLIAM CLOWES AND SONS, LIMITED, STAMFORD STREET AND CHARING CROSS.